ESTIMATION OF SALIVARY C REACTIVE PROTEIN IN PREGNANT WOMEN WITH OBESITY AND PERIODONTITIS AND THEIR POTENTIAL RISK FOR PRETERM BIRTH

A Dissertation submitted in

partialfulfillment of the requirements

for the degree of

MASTER OF DENTAL SURGERY

BRANCH – II

PERIODONTOLOGY

THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI – 600032

2016 – 2019

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I hereby declare that this dissertation titled " Estimation of salivary c reactive protein in pregnant women with obesity and periodontitis and their potential risk for preterm birth" is a bonafide and genuine research work carried out by me under the guidance of **Dr.C.S.PRABHAHAR, M.D.S., Professor, Head of the Department,** Department Of Periodontology, Best Dental Science College, Madurai – 625104.

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"Man needs difficulties in life because they are necessary to enjoy happiness"- Dr.A.P.J.Abdul Kalam

Good things come to those who believe, better things come to those who are patient and the best thing comes to those who never give up.

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At last everything comes in place only with the almighty blessings and my prayer to **sai** never ends.

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This agreement herein after the "Agreement" is entered into on this day31 Dec 2018, between the Best Dental Science College represented by its **Principal** having address at Best Dental Science College, Madurai – 625104, (hereafter referred to as, 'the College')

And

Mr.Dr.C.S.PRABHAHAR, M.D.S., aged 45 years working as **Professor and HOD** in Department of Periodontology at the College, having residence address at 9/3 Vallalar Street, Alagappapuram, Karaikudi – 630 002, Sivagangai district.

(herein after referred to as the 'Principal Investigator')

And

Miss. Dr. T. SUGANYA HARSHNI aged 25 years currently studying as **Post Graduate student** in Department of Periodontology, Best Dental College, Madurai-625104 (herein after referred to as the 'PG/Research student and co-investigator')

Whereas the PG/Research student as part of his curriculum undertakes to research on "ESTIMATION OF SALIVARY C REACTIVE PROTEIN IN PREGNANT WOMEN WITH OBESITY AND PERIODONTITIS AND THEIR POTENTIAL RISK FOR PRETERM BIRTH" for which purpose PG/Principal Investigator shall act as Principal Investigator and the college shall provide the requisite infrastructure based on availability and also provide facility to the PG/Research student as to the extent possible as a Co-investigator.

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LIST OF ABBREVIATIONS USED

APO	**ADVERSE PREGNANCY OUTCOMES**
ANOVA	ANALYSIS OF VARIANCE
BMI	BODY MASS INDEX
CAL	CLINICAL ATTACHMENT LEVEL
CEJ	CEMENTO ENAMEL JUNCTION
CRP	C REACTIVE PROTEIN
GCF	GINGIVAL CREVICULAR FLUID
GI	GINGIVAL INDEX
HCA	HISTOLOGICAL CHORIOAMINITIS
IgM	IMMUNOGLOBULIN M
IL-1β	INTERLEUKIN - 1β
IL-α	INTERLEUKIN - 1α
IUGR	INTRA UTERINE GROWTH RESTRICTION
Kg	KILOGRAM
LDL	LOW DENSITY LIPIDS
LBW	LOW BIRTH WEIGHT

LPS	LIPOPOLYSACCHARIDES
m	METRE
mm	MILLIMETRE
MMP	MATRIX METALLO PROTEINASE
PD	POCKET DEPTH
PE	PRE ECLAMPSIA
PG E_2	PROSTAGLANDIN E_2
PLBW	PRETERM LOW BIRTH WEIGHT
PTB	PRETERM BIRTH
PTD	PRETERM DELIVERY
TIMP	TISSUE INHIBITORS OF METALLO PROTEINASES
VLBW	VERY LOW BIRTH WEIGHT
WC	WAIST CIRCUMFERENCE

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Periodontal medicine, as suggested by **Offenbacher** , as a broad term that defines a rapidly emerging branch of periodontology focusing on the wealth of new data establishing a strong relationship between periodontal health or disease and systemic health or disease. It means a two-way relationship in which periodontal disease in an individual may be a powerful influence on an individual's systemic health or disease as well as the more customarily understood role that systemic disease may have influence on individual's periodontal health or disease [1] .

There are various modern pathogenic concepts for systemic disorders, which includes like auto intoxification, focal infection, psychosomatic disease, and autoimmunity. "Focal infection theory" was given by **Miller**, speak about the role of various oral pathogens in the systemic disease. There has been significant progress in validating the plausibility of oral disease disseminating to cause systemic diseases. Various studies have shown a substantial relation between poor oral hygiene to increased susceptibility for various systemic disorders. The field of periodontal medicine has evolved into a significant oral and systemic health care media, since its inception in 1996 during the world workshop in Periodontics [2] .

Periodontal disease is an inflammatory process, they affect the protective and supportive tissues found around the tooth. Bacterial plaque accumulation found on the tooth surface leads to the marginal tissue inflammation, known as gingivitis. When gingivitis is left untreated, it may progress to periodontitis, which is characterized by loss of periodontal attachment support [3].

Periodontal disease during pregnancy could be a risk factor for adverse pregnancy outcomes such as preterm birth. Women with the active periodontal disease during pregnancy might experience translocation of periopathogenic bacteria to the uteroplacental unit, which ultimately damages the placenta. Obesity has been suggested to be associated with periodontitis. Pre-pregnancy obesity could be an important and potentially modifiable source of preterm birth (PTB). Obese women are likely to have a higher risk for pregnancy outcomes. Because obesity has effects on metabolic and immune parameters, it may increase the host's susceptibility to periodontal disease. This association could be identified more clearly in pregnant women as pregnancy increases the host's susceptibility to systemic inflammation caused by pregnancy related hormonal influences on the immune system . Considering the abundance of evidence supporting the association among obesity, periodontitis, and PTB, it can be inferred that obesity and periodontitis before or during pregnancy have synergistic effects on PTB [4].

Preterm birth is the leading perinatal problems worldwide and has evident public health implications because it is closely related to perinatal mortality and morbidity. Multiple factors, some of which are preventable, have been associated with PTB , e.g., alcohol, smoking or drug use during pregnancy, high or low maternal age, low socioeconomic status, reduced prenatal care, low maternal body mass index (BMI), hypertension, generalized infections, genitourinary tract infections, cervical incompetence, diabetes, nutritional status, stress and multiple pregnancies. Increasing efforts have been made to diminish the effects of these risk factors by preventive interventions during prenatal care [5].

In **1891, Miller** published the theory of **"focal infection"** which hypothesized that oral foci of infection were responsible for a number of regional and systemic diseases, such as tonsillitis, pneumonia, endocarditis and septicemia. However, the lack of scientific evidence condemned this theory to dormancy. In **1990s, Offenbacher's** group studied about a bacteremia and "chamber" model to mimic a focal infection on pregnant hamsters found that periodontal bacteria and inflammatory mediators have the ability to disseminate systematically to the foetal-placental unit, via the blood circulation and which induces the pregnancy complications and they proposed oral infection, such as periodontitis, act as a distant infectious reservoir and affects the pregnancy outcome. During pregnancy, due to physiological hormonal changes, there is a systematic inclination of risk to periodontal disease.

A large number of studies associate an increase in the levels of local and systemic markers of inflammation with adverse pregnancy outcomes. Hence, the elevated levels of Interleukin-1β, Interleukin -α, Tumor Necrosis Factor -α , Prostaglandins E2, Fibronectin and α-Foetoprotein in the amniotic fluid have been associated with Preterm Birth , while other biomarkers such as MMPs, estriol, elastase, protease, phospholipase, prolactin, myeloperoxidase and tissue inhibitor of MMP (TIMP)-1 have been evaluated but with inconclusive results. Maternal serum increased with the levels of pro-inflammatory cytokines, such as IL-1, IL-6, IL-8 and TNF-α, has also been shown to be associated with prematurity or low birth weight. [4] C-reactive protein (CRP), is an acute phase reactant synthesized by the liver in response to pro-inflammatory cytokines, and are implicated as marker of systemic inflammation found to be associated with PTB. Besides PTB, elevated levels of CRP have been shown to be associated with an increased

risk for other pregnancy complications, such as intrauterine growth restriction (IUGR) and pre-eclampsia [6].

CRP increases the inflammatory response through complement activation, tissue damage, and induces the inflammatory cytokines and may mediate relationship between periodontitis and adverse pregnancy outcomes [7]. The presence of the biomarker CRP in saliva provides an opportunity for development of novel approaches for noninvasive assessment of inflammation [8].

The most of the physiological and hormonal changes in the life of a woman occurs during the pregnancy, and among that oral cavity is one of the targeted area involved by this changes. All functions of the mother's host should adapt to the new condition. Although other hormonal changes also found to occur, the most hormonal change is the increased production of estrogens and progesterone. The production of these hormones which gradually increases during the pregnancy until the 8th month. During the period of last month of the pregnancy, progesterone concentrations found to be remain relatively constant, whereas estrogen levels found to continue to rise. The reason for the increased production of hormones during pregnancy is mainly due to the placenta, which is involved over the production of progesterone and estrogens in early period of the pregnancy time , which is the the main source of these hormones from the 2nd trimester to term. Estrogen levels are found to rise more than 100-fold from the beginning period of pregnancy. Progesterone concentrations found to rise even more. The estrogen to progesterone ratio in the blood changes from 100:1 in early pregnancy to nearly it becomes 1:1 at term. During labor, when the placenta is withdrawn, there is marked fall occurs in both the progesterone and estrogens levels. Within 2–3 days of

delivery, the hormone concentrations will reaches their non-pregnant levels [9]. Maternal health has long been recognized as an important determinant in reducing the risk for pregnancy-related complications such as preterm birth and pre-eclampsia. The oral changes that can occur in pregnancy have been followed with focus of interest for many years. Physiological changes which occur in pregnant women adversely affect oral health status. Increase in the level of estrogen and progesterone enhances the inflammatory response and consequently brings changes in the gingival tissue. During pregnancy, the incidences of gingivitis and periodontitis are increased, and many pregnant women suffer from bleeding and swollen gums. Periodontal disease is a persistent bacterial infection, leads to a chronic and systemic challenge with the bacterial substances and host-derived inflammatory mediators that are capable of initiating and promoting systemic diseases. According to **Lieff et al., 2004,** adequate oral hygiene habits were mandatory to control the development of periopathogenic oral biofilms, which have been reported to be associated with poor obstetric outcomes [10]. Even though there exists in a controversy regarding the role of oral health as an independent contributor to abnormal pregnancy outcomes, the recognition and understanding of the importance of oral health has led to significant research into the role of maternal oral health in pregnancy outcomes [11].

AIM :

The aim of the present study is to estimate the level of salivary c reactive protein in pregnant women with obesity and periodontitis and to identify their potential risk for preterm birth.

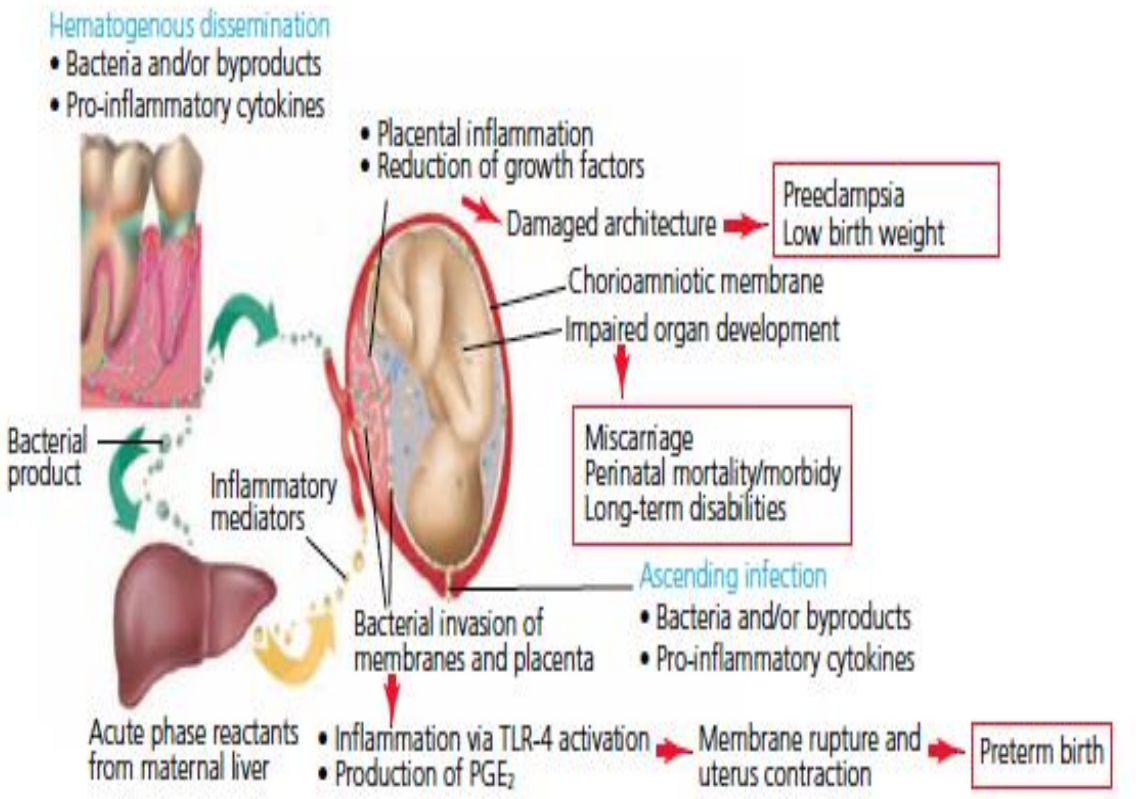
OBJECTIVES :

- To estimate the salivary c reactive protein in pregnant women and their correlation with preterm birth.
- To correlate the association between obesity, periodontitis and preterm birth.

GENERAL REVIEW

According to ***sir William Osler*** (1875) mouth is the mirror and gateway of the body, which reflects manifestations of other systemic diseases. Pregnancy represents a feto-maternal balance between the tumor like acquirement of the growing fetus and the maternal immune system that has the potential to reject the developing fetus. The placenta functions as a physiological buffer between the mother and the fetus to suppress maternal immune surveillance and to simultaneously exchange the nutrients and waste products needed for fetal growth. Maternal hormones, such as progesterone induce a relative state of mild immune tolerance to prevent rejection of the semi allogenic fetal graft, but the cellular and molecular inflammatory mechanism of rejection would remain intact. If this inflammatory process is regulated and selectively activated, it can provide an efficient mechanism for inducing delivery. The fact is that intra amniotic levels of many inflammatory mediators, such as PGE_2 increase during pregnancy and peak at delivery suggests that these mechanisms play an important role in normal physiologic parturition. Furthermore, if maternal infection threatens the fetus and the mother, then these mechanisms need to be functional as a safe guard to induce abortion to assure the survival of the mother. Thus, the maternal inflammatory response can be regarded as the effector arm of the immune response, which can be carefully orchestrated to regulate tissue responses leading to delivery, especially coordinating changes in cervical tone, membrane integrity, and uterine contraction. Thus, this common effector pathway of inflammation comes to bear in both normal physiologic parturition and under circumstances of pathology, especially when associated with early cervical dilation and effacement, preterm rupture of membranes, and premature uterine contraction [12].

FIGURE 1 : RELATIONSHIP BETWEEN PERIODONTITIS AND PRETERM BIRTH



Periodontitis is caused by a chronic, anaerobic gram-negative infection, but the severity of the disease appears to be principally driven by the magnitude of the host inflammatory response [13]. Bacterial Vaginosis (BV) is also a common infectious cause of preterm deliveries; however the maternal inflammatory response is the effector mechanism that leads to prematurity [14,15]. Recent data [16] suggest that more severe periodontal disease was found in PLBW mothers as compared to normal birth weight (NBW) mothers, even after adjusting for several known factors.

The possibility that periodontal gram negative infections may be important with respect to preterm birth has come from a study [17] in which periodontal disease was shown to be a significant risk for preterm birth. The condition of increased gingival inflammation during pregnancy [18] has been known for many years. In fact, there have been suggestions that it may be related to immunosuppression in the second trimester. The infected periodontium can also be regarded as a reservoir for both microbial products and inflammatory mediators.

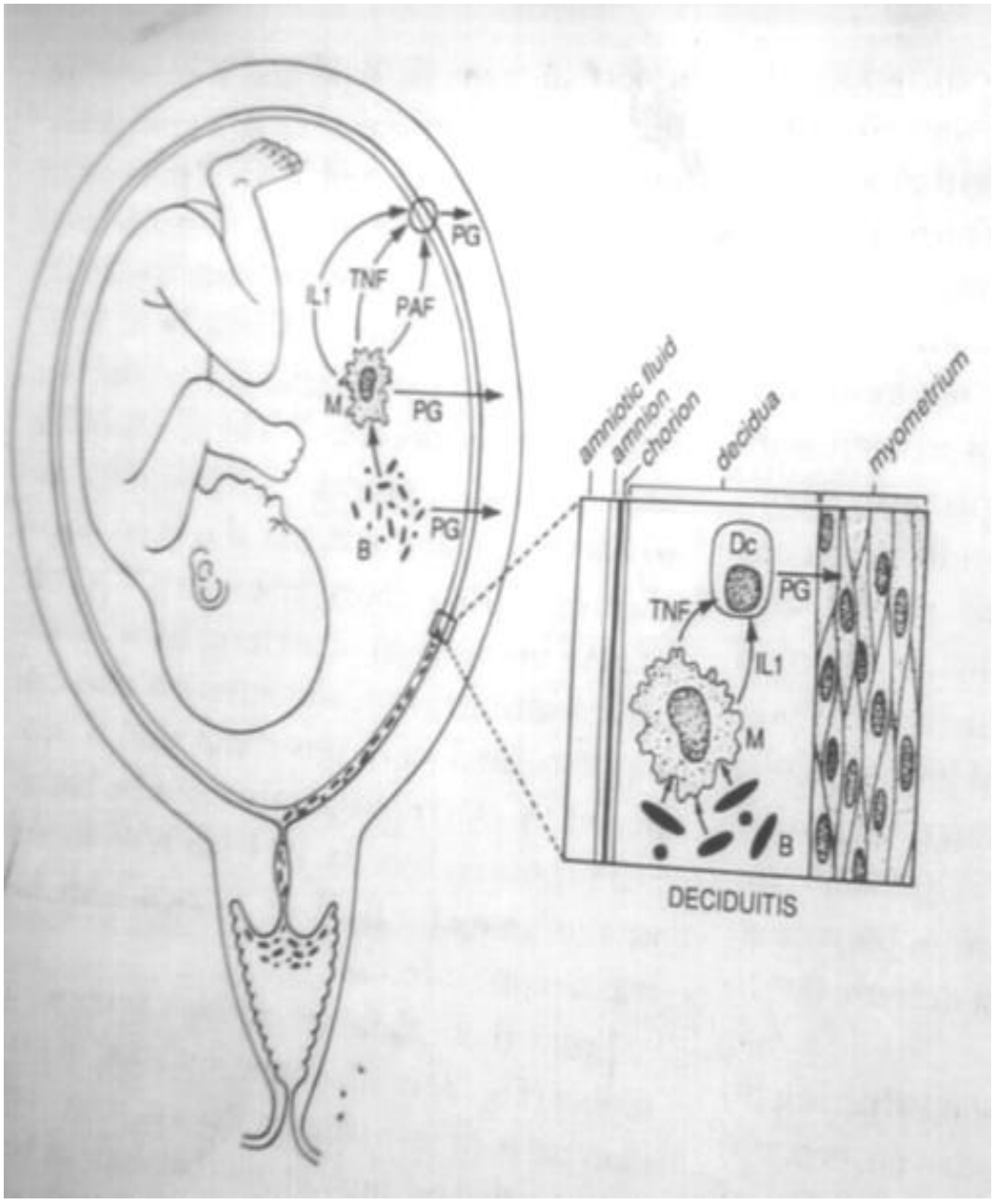
ASSOCIATION BETWEEN INFECTION AND PRETERM LOW BIRTH WEIGHT :

Several studies [19, 20, 21, 22, 23, 24] had demonstrated that there were association between infection and PLBW. The first evidence regarding this association was involved in the increased prevalence rate of maternal lower genitourinary tract infections with pregnancy complications such as PTL (Pre Term Labor) and LBW (Low Birth Weight). Rates of this preterm delivery were 1.5 to 2.3 time's normal than those have been found among the women with group B *Streptococci bacteriuria*. Gram negative *Bacteroides* species also been investigated to find their association with preterm delivery and premature rupture of membrane. One study [22] showed that 40% increase in preterm delivery rates in mothers who were found to colonized with cervical *Bacteroides* at their initial prenatal visits. Vaginal *Bacteroides* colonization was found to be associated with 60% increased risk of preterm delivery.

Furthermore, genitourinary tract infection also found to be associated with the LBW, without infection of the fetal placental unit. These observations have supported the current opinion that preterm LBW that occurs as a result of infection is mediated

indirectly, mainly by the translocation of bacterial products such as endotoxin (lipopolysaccharides, LPS) and also by the action of maternally produced inflammatory mediators. TNF -α level also shown to be increased in periodontal disease conditions.

FIGURE 2: ROLE OF INFLAMMATORY MEDIATORS ON PRETERM LABOR



Cellular and biochemicalmechanisms involved in the initiation of preterm labor in case of intrauterine infection

(B = bacteria;

M = monocyte / macrophage; Dc = decidua;

IL1 = interleukin – 1;

TNF = tumor necrosis factor / cachectin;

PG = prostaglandin;

PAF = platelet activating factor).

The inflammation and intra amniotic link about levels of cytokines has been recently examined in a study [25] conducted at the University of North Carolina. In investigation about the levels of PGE_2 and IL-1 present within the gingival crevicular fluid (GCF) of 18 women in sample of early mid-trimester were compared to the levels of these mediators in amniotic fluid. The study demonstrated that the GCF levels of PGE_2 and IL-l were highly correlated with the levels of intra amniotic PGE_2 and IL-l, and it appears that the GCF levels may eventually be used as a diagnostic test to provide an indirect estimate of the amniotic fluid levels of PGE_2 , appropriate studies were conducted to establish about sensitivity and specificity. This may provide information about the possible screening of expectant mothers by measuring GCF levels of PGE_2 and IL-l , which are known amniotic factors associated with PLBW. It is a far less

invasive procedure when compared to amniocentesis and has a reduced risk of affecting the developing fetus.

RELATIONSHIP BETWEEN PERIODONTITIS AND OBESITY:

T.Saito et al (2001) [26], studied the relationship between upper body obesity and periodontitis. They did the study among 643 apparently healthy, dentulous Japanese adults. Waist-hip ratio, body-mass index (BMI), and body fat were considered as significant risk indicators for periodontitis. Subjects were divided into four BMI categories. In only the subjects with high waist-hip ratio, higher categories of BMI significantly increased the adjusted risk of periodontitis, when compared with the subjects with low waist-hip ratios and the lowest category of BMI.

Jorge A. Zermeño-Ibarra et al (2010) [27] conducted a cross sectional study to know about the relationship between overweight-obesity and periodontal disease in Mexico individuals. They found that periodontal disease showed statistically significant differences in the group of subjects with overweight-obesity.

Saxlin T et al (2010) [28] performed a longitudinal study to investigate the association between the body weight and periodontal infection. He concluded that this longitudinal study does not provide any evidence that overweight and obesity can be considered as significant risk factors in the pathogenesis of periodontal infection.

Chaffee BW et al. (2010) [29] done a systematic review of 70 cross sectional studies, suggested that 41 studies showed positive association between chronic periodontal disease and obesity. Also positive association between periodontal disease and obesity was suggested across diverse populations. The prevalence of periodontal disease is likely to be higher among obese patients, although there was no current evidence to recommend differences in treatment planning.

Singh MP et al. (2013) [30] did a study to evaluate the relationship between obesity and periodontitis and showed that the prevalence of periodontitis were significantly more in obese patients than in non obese. They concluded that strong correlation exists between obesity& periodontitis. Obese with high serum triglycerides & LDL could be at higher risk of periodontitis.

Palle AR et al. (2013) [31] demonstrated significant association between measures of overall and abdominal obesity (BMI and WC) and periodontal disease showed significant association in their cross sectional study.

Giri DK et al. (2013) [32] did a study to determine the association of obesity with periodontal disease in a semi urban Indian population. No association was obtained between BMI and periodontitis. Hence it was concluded, that good oral hygiene and normal body weight can reduce the overall inflammatory burden, thereby reducing the risk for development of periodontal disease.

L. Scorzetti et al (2013) [33] studied the relationship between the obesity and periodontal disease in children. The study showed an association between obesity and periodontal risk indicators in children that in the long term may lead to oral conditions. And they concluded that the oral health of overweight/obese subjects should be more carefully supervised and checked in order to prevent oral alterations.

Jelena Prpic et al (2013) [34] studied the relationship between the association of obesity with periodontitis, tooth loss and oral hygiene in non-smoking adults. They concluded that obesity was associated with tooth loss, oral hygiene, and education level in the investigated group and the study showed that BMI could not be correlated with severity of periodontal disease, except in poorly educated women aged 36–55 years.

Shashikanth Hegde et al (2014) [35] conducted a pilot study to know the role of obesity and chronic periodontitis . A total of 20 systemically healthy patients, aged 30 to 60 years were included in the study. Gingival index was significant in obese group with chronic periodontitis compared to non obese group with chronic periodontitis. This pilot study had shown the significant differences in gingival index, waist circumference, body mass index, total cholesterol, triglycerides and LDL levels, between obese and non obese group. The probing depth and loss of attachment were higher in the obese group, when compared to the non obese group.

Amelie keller et al (2015) [36] analysed among eight longitudinal and five intervention studies and suggested that overweight, obesity, weight gain, and increased waist circumference may act as a risk factors for the development of periodontitis or results in worsening of periodontal measures.

Sania et al (2017) [37] reviewed about the relationship between obesity and periodontitis concluded that this relationship needs further investigation, periodontists should counsel obese persons regarding the possible oral complications of obesity, to diminish the future risk of occurrence for these individuals.

Jiabao Sun BA et al. (2017) [38] analyzed and done literature search that obesity and periodontal disease can exert similar pathogenic effects via common pathways, and can influence each other bidirectionally. Elucidating about the relationship between obesity and periodontal disease allows for the development of care guidelines and recommendations for clinicians and the public.

RELATIONSHIP BETWEEN PERIODONTITIS AND ADVERSE OUTCOMES DURING PREGNANCY:

S.Offenbacher et al (2001) [39] conducted a 5 year prospective study designed to determine whether maternal periodontal disease contributes to the risk of prematurity and growth restriction in presence of tradional obstetric risk factors . The findings indicate that periodontal disease incidence or progression has a significant effect on birth weight and maternal periodontal infection affects fetal growth.

Juha Oittinen et al (2005) [40] studied clinically about Periodontal disease and bacterial vaginosis to increase the risk for adverse pregnancy outcome among 252 women. This study suggested that pre-pregnancy counseling should include both oral and vaginal examinations to rule out periodontal disease and bacterial Vaginosis. These ultimately have an impact on antenatal healthcare, and decrease the risk for adverse pregnancy outcome.

A.Contreras et al (2006) [41] conducted a case control study in pregnant women to know whether Periodontitis is associated with pre-eclampsia in Pregnant Women. They concluded that chronic periodontal disease and the presence of *P. gingivalis, T. forsythensis, and E. corrodens* were found to be significantly associated with preeclampsia in pregnant women.

Fouzia Tarannum et al (2007) [42] studied the effect of periodontal Therapy on Pregnancy Outcome in Women Affected by Periodontitis. They concluded that Non-surgical periodontal therapy can reduce the risk for preterm births in mothers who are affected by

periodontitis. Additional multicentered, randomized, controlled clinical trials are required to confirm this link between periodontitis and preterm low birth weight.

Pitiphat W et al (2008) [43] conducted a prospective study to know the adverse outcomes of maternal periodontitis. Within their limitations of the study, their results suggested that periodontitis is an independent risk factor for poor pregnancy outcome among middle-class women.

M. V. Vettore et al (2008) [44] studied the relationship between periodontal disease on preterm low birth weight. The results were that the mean periodontal pocket depth was significantly higher in non preterm low-birth weight controls than in subjects in the preterm low birth weight, preterm and/or low birth weight, and preterm and low-birth weight groups. Clinical attachment level measures were not different between all pairs of cases and control groups. Groups did not differ with respect to the mean proportions of different microbial complexes. The mean counts of *Treponema socranskii* were lower in all case groups compared with the control group.

Agueda A et al (2008) [45] conducted a prospective cohort study to analyse that periodontal disease as a risk factor for adverse pregnancy outcomes . This study found a moderate association between periodontitis and PB. Further research is required to establish whether periodontitis is a risk factor for PB and/or LBW.

Bryan S. Michalowicz et al (2009) [46] conducted a study to determine serum inflammatory mediators in pregnancy and their changes after periodontal treatment and association with pregnancy outcomes. A total of 823 pregnant women were included in study. They concluded that non-surgical mechanical periodontal treatment in pregnant women, delivered before 21 weeks of gestation, did not reduce systemic (serum) markers

of inflammation. In pregnant women with periodontitis, levels of these markers at 13 to 17 weeks and 29 to 32 weeks of gestation were not associated with infant birth weight or a risk for preterm birth.

Nabet C, Lelong N et al (2010) [47] analyzed the association between maternal periodontitis and preterm birth (≥37 weeks' gestation) according to the causes of preterm birth. They concluded that maternal periodontitis is associated with an increased risk of induced preterm birth due to pre-eclampsia.

D. Gandhimadhi, and R. Mythili (2010) [48] conducted study to know about Periodontal infection as a risk factor for preterm low birth weight. In this study the cases had significantly more attachment loss and probing pocket depth, poor oral hygiene, more percentage of sites with attachment loss (Extent) and more mean attachment loss per site (Severity) and less Hb than controls. The number of visits for prenatal care and the percentage of sites with CAL≥2mm (Extent 2) remained significant when compared to other variables. The study indicated that periodontal disease act as a contributing factor for the preterm low birth weight condition .

Sapna singh et al (2011) [49] did a study to assess whether periodontal disease is predictive of premature gestation in an identified population of women at risk for birth complications as a consequence of medical factors. This research demonstrated that periodontal disease in normal pregnant women is significantly associated with decreased infant birth weight, providing new evidence on the relationship between periodontal disease and birth weight. Nevertheless, the association between periodontal disease and birth weight infant should be further explored in new observational and intervention

studies to establish whether it is casual or incidental, and to generalize the findings in diverse populations.

Guimaraes AN et al (2012) [50] aimed to study the very low and low birth weight association with maternal periodontitis. They concluded that maternal periodontitis was associated with a decrease in mean birth weight, as well as with LBW and VLBW.

Carrillo-de-Albornoz A et al (2012) [51] studied the gingival changes during pregnancy. The results were that PlI was the strongest predictor implicated in the GI throughout pregnancy and after delivery. In second and third trimesters the presence of *Porphyromonas gingivalis* significantly found to contribute to the worsening of gingival inflammation. When they are compared to the non-pregnant group, significant differences were found in TNF-α amounts and concentrations and in the third trimester site-specific GI.

Pourandokht Afshari et al (2013) [52] conducted a study among 180 pregnant women to relate the maternal periodontitis and adverse pregnancy outcomes which results indicate in development and severity of periodontitis leads to increase risk of adverse pregnancy outcomes.

Yiqiong Xie et al (2013) [53] conducted a follow up study among 39 females about change of periodontal status before and after pregnancy. They concluded that pregnancy may be associated with an increased risk of periodontal disease. The association is not different between females with Gestational Diabetes Mellitus and females without Gestational Diabetes Mellitus during pregnancy.

Madianos PN et al (2013) [6] evaluated the evidence on potential biological pathways underlying the possible association between periodontal disease and adverse pregnancy

outcomes (APOs). The evaluation resulted that periodontal pathogens/byproducts may reach the placenta and spread to the foetal circulation and amniotic fluid. Their presence in the foeto-placental compartment stimulated a foetal immune/inflammatory response, which are characterized by the production of IgM antibodies against the pathogens and the secretion of increased levels of inflammatory mediators, which in turn may cause miscarriage or premature birth.

Mansi Bansal et al (2013) [54] evaluated the relationship between maternal periodontal status and preterm low birth weight. They concluded that many common risk factors for PLBW are present along with periodontal diseases (eg, age, socioeconomic status, and smoking). However, because the inflammatory mediators that occur in periodontal diseases also play an important part in the initiation of labor, there can be a possible biologic mechanism that could link the two conditions.

Mahmoud Edessy et al (2014) [7] evaluated the relationship between periodontal diseases and adverse pregnancy outcomes among 300 patients. They found a significant relationship between them.

Pushpalatha Govindaraju et al (2015) [55] conducted a case control study to know the relationship between maternal periodontal disease and preterm birth. They concluded that an observable relationship was noticed between periodontitis and gestational age, and a positive correlation was found with respect to PTB and periodontitis. Further studies should be designed to establish periodontal disease as an independent risk factor for PTB/preterm low birth weight.

C REACTIVE PROTEIN :

CRP is a protein found in plasma involved in the acute phase response. CRP was first discovered in 1930 as a protein that could precipitate the C polysaccharide derived from the Pneumococcal cell wall (Tillet and Francis 1930). CRP is a interest of biomarker in various disease and now a days is a indicator of disease activity . CRP consists of five identical, noncovalently associated 23kDa protomers arranged symmetrically around a central pore. The term "pentraxins" has been used to describe the family of related proteins with this structure. In humans, plasma levels of CRP may rise rapidly and markedly, as much as 1000-fold or more, after an acute inflammatory stimulus, largely reflecting increased synthesis by hepatocytes. CRP induction is part of a larger picture of reorchestration of liver gene expression during inflammatory states, the acute phase response, in which synthesis of many plasma proteins is increased [56] .

Waranuch Pitiphatet al (2005) [57] studied about the C reactive protein in early pregnancy and preterm delivery . In summary, they found that CRP levels of more than 8 mg/liter in early pregnancy were associated with preterm delivery independent of many other determinants of preterm delivery. The association was apparent primarily for spontaneous preterm delivery. These results were consistent with the hypothesis that chronic low-grade inflammation may raise CRP levels and cause preterm delivery.

Waranuch Pitiphat et al (2007) [58] studied about periodontitis and plasma c reactive protein during pregnancy . Their findings suggest that periodontitis may increase CRP levels in pregnancy. CRP could potentially mediate the association of periodontitis with adverse pregnancy outcomes.

Adriana R. Brasil et al (2007) [59] studied to elicit that C-reactive protein as an indicator of low intensity inflammation in children and adolescents with and without obesity. The objectives is to determine the levels of high sensitivity C-reactive protein (hsCRP) in children/adolescents with and without obesity and their correlation with body mass index (BMI) and clinical and laboratory variables. They concluded that the hsCRP concentrations increased as BMI increased. The majority of individuals who were not overweight exhibited hsCRP concentrations of less than 2 mg/L.

Anupriya Sharma et al (2009) [60] evaluated the plasma C-reactive protein levels in pregnant women with and without periodontal disease as a comparative study . The findings from the study suggested that periodontal disease in pregnant women is associated with increased C- reactive protein levels in early pregnancy, incidence of preterm delivery is higher in pregnant women with periodontal disease compared to healthy controls, periodontal therapy during pregnancy reduces plasma CRP levels and there is decrease in incidence of preterm delivery after periodontal therapy.

Mahdieh Shojaee et al (2013) [61] aimed to study the C - Reactive Protein levels in patients with periodontal disease and normal Subjects. The results of this study showed a significant correlation between salivary CRP levels and severity of periodontal disease. Their study showed that the measurement of salivary CRP can be used as a non-invasive and reliable test for the detection and screening of periodontal disease in healthy people.

Bertha L. Bullen et al (2013) [62] studied the association between maternal C-reactive protein (CRP) and preterm delivery (PTD) pathways, CRP was measured in maternal plasma collected at mid-pregnancy. They find that plasma CRP measured at mid-

pregnancy is associated with sPTD (spontaneous preterm delivery) and that the presence of HCA(histologic chorioamnionitis) at delivery accounts for much of this association. However, a significant association between CRP and sPTD in the absence of HCA remains in higher BMI women, suggesting that CRP in overweight/obese women may mark activation of pathways to PTD that are distinct from pathways related to HCA.

Ajay halder et al (2013) [63] evaluated the predictive significance of C- reactive protein in spontaneous preterm delivery. A group of subject with 280 pregnant women , found between 12-22 weeks of gestational age attending antenatal clinic were included in a prospective cohort and followed through the pregnancy, delivery and early puerperium. CRP positivity in early pregnancy is associated with nearly a two fold increased risk of preterm delivery. Complications of neonates like preterm birth , low birth weight, septicaemia, birth asphyxia and many others are more common in CRP positive mothers.

Deepika Jayaprakash et al (2014) [64] studied the Effect of periodontal therapy on C-reactive protein levels in gingival crevicular fluid of patients with gingivitis and chronic periodontitis. Within the limitations of this study, they concluded that GCF CRP level progressively increases from periodontal health to disease. It can also be stated that there is a decrease in GCF CRP levels with periodontal treatment.

Rajiv Saini (2014) [65] studied whether periodontitis is associated with elevated levels of systemic inflammatory biomarker c reactive protein . This study is outlined to correlate salivary c reactive protein level in health, gingivitis, and periodontitis which assess their values after completing SRP. They concluded that a correlation exists between the periodontal disease and CRP. As the periodontal disease regresses the value of CRP significantly gets down. SRP was effective in reducing the level of CRP.

Stepan Podzimek et al (2015) [66] , the aim of this study was to compare and evaluate the systemic levels of CRP in the peripheral blood samples of patients found with chronic periodontitis and aggressive periodontitis, gingivitis, and gingival recessions and compare them with other periodontal clinical parameters. Their study results show that CRP levels increase subsequently with the severity of the periodontal disease. The lowest CRP levels were found in patients with gingival recessions, increasing in patients with gingivitis and patients with chronic periodontitis, with the highest levels found in aggressive periodontitis patients. The bleeding on probing index showed much better positive correlation with the CRP levels compared to the pocket depth index in both periodontitis patient groups, especially in aggressive periodontitis patients.

P.Savitha et al (2015) [67] compared the salivary CRP level in chronic periodontitis and healthy individual. The results of this present study showed that salivary CRP concentrations increased in patients with periodontitis comparing to the healthy individual and confirming the theory that salivary CRP is increased in inflammatory conditions. CRP level in saliva was demonstrable in periodontitis cases. In normal individual the CRP level is 0.1 and less than 0.1 mg/L where as in periodontitis cases, 60% of the patients have shown 0.2 mg/L. So, the salivary CRP level demonstration is definitely having diagnostic value and prognostic value and it can be used as a tool to access the improvement in the patient's condition in periodontal diseases.

Padmakanth Mannava et al (2016) [68] comparatively elevated the c reactive proteins in pregnant women with and without periodontal pathologies . They concluded that casual association might exist between the CRP levels and periodontal disease in pregnant women and the levels may also get elevated in pregnant women.

The study was approved by the Scientific and Ethical Committee review board of Best Dental Science College and Hospital, Madurai. The study was carried out from January 2017 to October 2017.

STUDY POPULATION

- The study population were recruited from patients attending outpatient clinics of private clinic in Madurai. The study was explained to the concern Gynaecologist and Obstetrician in the clinic and permission was obtained to conduct the study.
- A total of 74 subjects (pregnant women) were included in the study. Out of which 6 patients were excluded from study, due to loss of follow up. Finally 68 subjects were included .
- All the participants in the study were verbally informed about the nature, risks and benefits of the study and a written informed consent was obtained.

CRITERIA FOR SELECTION OF SUBJECTS

Inclusion Criteria:

- Pregnant women with age between 19 years and 40 years.
- Gestational age ≤ 32 weeks
- Singleton pregnancy [1]

Exclusion criteria:

- Maternal age below 18 and above 40 years .
- Women with multifoetal pregnancy due to greater risk of preterm birth .[1,2]
- Uterine or cervical anomalies.
- Any previous history of preterm.

- Chronic pre gestational conditions that affect pregnancy outcomes, such as pre gestational diabetes and chronic hypertension. [1]
- Presence of less than 20 teeth.
- Alcohol or drug abuse. [1]

ARMAMENTARIUM

Armamentarium for clinical evaluation:

- Mouth mirror
- Explorer
- Periodontal Williams probe
- Height chart

Armamentarium for saliva sample collection:

- Sample collecting sterile container

Armamentarium for transport of collected saliva:

- Ice packs within thermocole box

CLINICAL PARAMETERS:

- Gingival index (GI) [69]
- Probing pocket depth (PPD) [70]
- Clinical attachment loss (CAL) [2]
- Body mass index (BMI) [70]

Gingival Index (Loe and Silness 1963)

Table 1: Gingival Index Scores

Score	Criteria
0	Absence of inflammation/normal gingival
1	Mild inflammation, slight change in colour, slight edema and no bleeding on probing
2	Moderate inflammation, moderate glazing, redness, edema, hypertrophy and bleeding on probing.
3	Severe inflammation, marked redness, hypertrophy, ulceration and tendency for spontaneous bleeding.

CALCULATION

- *GI Score for the area:* Each area (disto-facial, facial, mesio-facial, lingual) is assigned a score from 0 to 3
- *GI Score for a tooth:* The scores from the four areas of the tooth are added and then divided by four.
- *GI score for the individual:* The indices for each of the teeth are added and then divided by the total number of teeth examined. The scores range from 0 to 3.

Table 2: Gingival Index Interpretation

Gingival scores	Condition
0.1-1.0	Mild Gingivitis
1.1-2.0	Moderate Gingivitis
2.1-3.0	Severe Gingivitis

Probing pocket depth:

Determined at six sites (mesio-buccal, mid-buccal, disto-buccal, disto-lingual, mid-lingual, and mesio-lingual surfaces) of all teeth except for the third molars. Measured from the gingival margin to the bottom of the gingival sulcus.

Clinical loss of attachment:

Determined at six sites (mesio-buccal, mid-buccal, disto-buccal, disto-lingual, mid-lingual, and mesio-lingual surfaces) of all teeth except for the third molars. Measured from the cemento-enamel junction (CEJ) to the bottom of the sulcus.

Body mass index:

TABLE 3 : WHO BMI cut-off points

BMI SCORE (kg / m^2)	CRITERIA
< 16	Severe underweight
16.0-16.9	Moderate underweight
17.0-18.49	Mild underweight
18.5-24.9	Normal range
≥25	Over weight
25-29.9	Pre- obese
≥30.0	obesity
30-34.4	obese class I
35-39.9	Obese class II
≥40	Obese class III

Overweight and obesity were combined into a single category for analysis because of the small number of obese pregnant women. BMI was calculated depending on pre-pregnancy weight. The values are obtained from the patient through face to face interview [1]

DEMOGRAPHIC AND HEALTH INFORMATION: All patients responded to a demographic questionnaire that included pre-pregnancy weight and height for determination of body mass index (BMI), health behaviors. All of these parameters were

obtained from a face-to-face interview. Pre-pregnancy weight and height were self-reported. [1]

SALIVARY C REACTIVE PROTEIN COLLECTION AND ESTIMATION:

The saliva sample is collected before conducting the periodontal examination. After a rinse of the mouth with water, saliva was allowed to accumulate in the floor of the mouth for approximately 2 minutes and repeatedly expectorated into a sterile sample container to collect approximately 5 ml and then the sample is stored in a thermocole ice box till they are transported to laboratory. The samples are transported within 24 hours after collection.[71] The salivary CRP is evaluated by particle-enhanced turbidimetric immunoassay (CRP calibrator Euro diagnostic systems).

CLINICAL EXAMINATION:

Periodontal conditions were assessed by measuring the Gingival index, probing pocket depth and clinical loss of attachment. Periodontitis was defined as two or more inter-proximal sites with CAL ≥4 mm that were not on the same tooth (Page & Eke 2007). [72,73,1]

GROUPING OF THE PATIENT:

The samples were divided into four groups

Group A: neither obesity nor Periodontitis

Group B: Only Obesity

Group C: Only Periodontitis

Group D: Both Obesity and Periodontitis.

Each group had a sample of 17.

PREGNANCY OUTCOME:

After the patient delivered, data on the occurrence of PTB were obtained from obstetricians from hospital records. PTB was defined as delivery at <37 weeks [1]. Infants weighing less than 2,500 grams at birth are considered to be low birth weight.

FIGURE 3: ARMAMENTARIUM FOR CLINICAL EVALUATION

FIGURE 4 : MOUTH MIRROR AND WILLIAMS PROBE

FIGURE 5 : ARMAMENTARIUM FOR BODY MASS INDEX EVALUATION-HEIGHT CHART IN CENTIMETERS

FIGURE 6 : PARTICLE-ENHANCED TURBIDIMETRIC IMMUNOASSAY (CRP CALIBRATOR EURO DIAGNOSTIC SYSTEMS)

FIGURE 7: SALIVARY SAMPLE COLLECTION

FIGURE 8 : INTRA ORAL PHOTOGRAPH

FIGURE 9 : CLINICAL EXAMINATION OF PROBING POCKET DEPTH

FIGURE 10: PHOTOGRAPH OF DELIVERED INFANT

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LAB No.	LA-00689	Date	24/Jan/2017
Name	**Mrs. KALLADEVI**	Age / Sex	27Yrs / F
Ref.by	Dr. SUGANYAHARSHNI BDS.MDS.,		

SEROLOGY TEST

Test Name	Result	Units	Normal Range
SALIVA FOR CRP	4.8	mg/L	

----- Care for Ever -----

Lab Technologist
26/1/17

All Investigations have their limitations which are imposed by the limits of sensitivity and specificity of individual assay procedures as well as the quality of the specimen received by the laboratory/hospital report may vary depend on the technology. Value of two technologies are not comparable.

The information collected regarding all the selected cases were recorded in a master chart. Data analysis was done with the help of computer by using SPSS 16 software and Sigma stat 3.5 version. Using this software mean, standard deviation and 'p' value were calculated through One way ANOVA, Chi square test, Pearson correlation and P value of < 0.05 was taken as significant.

RESULTS

The present study was conducted in private hospital to estimate the level of salivary c reactive protein in pregnant women with obesity and periodontitis and to identify their potential risk for preterm birth. A total of 74 subjects satisfying the selection criteria were included in the study. Finally , due to loss in follow up of 6 subjects , 68 subjects were finally included in the study.

The clinical parameters including gingival index, probing pocket depth, clinical attachment loss, body mass index are evaluated and the salivary sample is collected and salivary c reactive protein is estimated. Then patients are followed up and post obstetric details are collected. The results thus obtained were tabulated and subjected to statistical software SPSS 16 and Sigma stat 3.5 version.

Comparison of mean age between group A, B, C and D (Table 1 & Graph 1)

Mean age value of group A, B, C & D was 23.06, 24.88, 26.12 and 24.53 respectively. There was no significant difference between the four group values. p value was 0.188 which was not significant.

Comparison of body mass index values (kg/m^2) between group A, B, C and D (Table 2 & Graph 2)

Mean body mass index values of group A, B, C & D was 21.52, 29.13, 22.49 and 30.37 respectively. There was highly significant difference between among four groups. p value was <0.001 Significant.

Comparison of Mean values of gingival index between the group A, B, C and D (Table 3 & Graph 3)

Mean value of gingival index in group A, B, C & D was 1.15, 1.02, 1.67 & 1.75 respectively. There was highly significant difference among the four groups. p value was <0.001 Significant. The mean gingival index values are found to be increased in group C and D.

Comparison of mean probing pocket depth (in mm) values between group A, B, C and D (Table 4 and Graph 4)

Mean value of probing pocket depth in Group A, B, C & D was 2.53, 2.65, 5.41 & 5.29 respectively. There was highly significant difference among the four groups. p value was <0.001 statistically significant. The probing pocket depth mean values are found to be increased in group C and D.

Comparison of mean clinical attachment (in mm) loss between group A, B, C and D (Table 5 and Graph 5)

Mean clinical attachment loss of group A, B, C & D was 2.59, 2.65, 5.35 & 5.41 respectively. There was highly significant difference among the four groups. p value was <0.001 statistically significant. The clinical attachment loss was increased in group C and D.

Mean salivary c reactive protein (mg/L) in group A, B, C and D (Table 6 and Graph 6)

Mean value of salivary c reactive protein between groups A, B, C & D was 0.17, 0.09, 1.32 & 0.60 respectively. The mean value was increased in group C and D, when compared to group A and B.

Correlation between salivary c reactive protein (mg/L) and clinical parameters of group A (Table 7)

Moderate correlation was found between GI and Salivary CRP (mg/L). Good correlation (0.710, 0.663) results were found between PPD, CAL and salivary CRP (mg/L).

Correlation between salivary c reactive protein (mg/L) and clinical parameters of group B (Table 8)

Moderate correlation was found between GI and Salivary CRP. Good correlation was (0.660, 0.648) found between PPD, CAL Vs salivary CRP. Therefore PPD and CAL increases simultaneously salivary CRP value was also increased.

Correlation between Salivary c reactive protein (mg/L) and clinical parameters of group C (Table 9)

Good correlation (0.751, 0.717) was found between GI and PPD and Salivary .Moderate correlation (0.470) was found between PPD, CAL and salivary CRP.

Correlation between Salivary CRP (mg/L) Vs Other parameters in Group D (Table 10)

Good correlation (0.749, 0.691, and 0.725) was found between GI, PPD, CAL Vs Salivary CRP.

Comparison of distribution level of salivary c reactive protein values between groups (Table 11 A) and p value comparison among group A to other groups (Table 11B)

- In group A, 12 cases are found with value <0.1 salivary CRP, when compare to group C only 6 cases are found with <0.1 salivary CRP value. There was significant difference between Group A Vs Group C. p value was 0.039, which was Significant.
- In group B, 14 cases are found with <0.1 salivary CRP value, when compare to group C only 6 cases are found with <0.1 salivary CRP value .There was significant difference between Group A Vs Group C. p value was 0.005, which was Significant.
- In group B, 14 cases are found with <0.1 salivary CRP value , when compare to group C only 6 cases are found with <0.1 salivary CRP value . There was significant difference between Group A Vs Group C. p value was 0.005, which was significant.

Distribution of delivery week of infant among the group A, B, C and D (Table 12 & Graph 7)

Significant difference was found between group A Vs group B and group A Vs group D. No significant difference between group B Vs group C and group C Vs group D.

Distribution of birth weight (kilogram) among the group A, B, C and D (Table 13 & Graph 8)

Significant difference was found between group A Vs group D. No significant difference between group A Vs group B and group A Vs group C was found.

Distribution of gestational week during sampling among the group A, B, C and D (Table 14 & Graph 9)

It was found that when group A compared to other groups, the p value was 0.670 which is not significant.

Correlation of delivery week, birth weight, gestational week with salivary CRP (mg/L) in group A (Table 15)

In Group A, there was negative moderate correlation between delivery week and birth weight to salivary CRP. But low correlation was evident between gestational week and salivary CRP.

Correlation of delivery week, birth weight, gestational week with salivary CRP (mg/L) in group B (Table 16)

In group B, there was very low correlation between delivery week and salivary CRP and low correlation between birth weight and salivary CRP, and gestational week and salivary CRP was 0.309 which is low correlation.

Correlation of delivery week, birth weight, gestational week with salivary CRP (mg/L) in group C (Table 17)

In group C, there was negative Good correlation between Delivery week and Salivary CRP, birth weight and salivary CRP. But very low correlation found between Gestational week and Salivary CRP.

Correlation of delivery week, birth weight, gestational week with salivary CRP (mg/L) in group D (Table 18)

In group D, there was negative good correlation between Delivery week and Salivary CRP , Birth weight and salivary CRP. But very low correlation was found between Gestational week and Salivary CRP was 0.132.

Odd ratio and confidence interval calculation between control group A and group B about low birth weight parameter. (Table 19)

The table explains that the odd ratio =1.00 with C.I =0.0574 to 17.4121.

Odd ratio and confidence interval calculation between control group A and group B about pre term birth parameter. (Table 20)

The table explains that the odd ratio =8.55 with C.I =1.7358 to 42.1700

Odd ratio and confidence interval calculation between control group A and group C about low birth weight parameter. (Table 21)

The table explains that the odd ratio = 15.75 with C.I = 1.67 to 148.12.

Odd ratio and confidence interval calculation between control group A and group C about pre term birth parameter. (Table 22)

The table explains that the odd ratio = 2.54 with C.I = 0.516 to 12.546 .

Odd ratio and confidence interval calculation between control group A and group D about pre term birth parameter. (Table 23)

The table explains that the odd ratio =11.00 with C.I = 1.1931 to 105.1363

Odd ratio and confidence interval calculation between control group A and group D about low birth weight parameter. (Table 24)

The table explains that the odd ratio = 8.5556 with C.I = 1.7358 to 42.1700

Relationship between salivary CRP (mg/L) and delivery weeks (Graph 10)

The graph explains about the relationship between salivary CRP and delivery weeks. In group C and D, when salivary CRP was >0.1 the mean delivery weeks was 35.2 and 35.8 respectively.

Relationship between salivary CRP (mg/L) and birth weight (in kilogram) (Graph 11)

The graph explains about the relationship between salivary CRP and birth weight. In group C and D, when salivary CRP is >0.1 the mean delivery weeks was 2.64 and 2.69 respectively.

Relationship between salivary CRP (mg/L) and gestational week (Graph 12)

The graph explains about the relationship between salivary CRP and gestational week. In group A and B there was low correlation , while in group C and D there was very low correlation.

Table – 1 Comparison of mean age between groups A, B, C and D.

Age	Group A	Group B	Group C	Group D
Mean	23.06	24.88	26.12	24.53
SD	3.59	4.28	4.4	3.86
p value	0.188 Not Significant			

Table – 2 Comparison of body mass index values (kg/m^2) between groups A, B, C and D

BMI	Group A	Group B	Group C	Group D
Mean	21.52	29.13	22.49	30.37
SD	1.96	3.35	1.63	3.91
p value	< 0.001 Significant			

Table – 3 Comparison of Mean values of gingival index between the group A, B, C and D

GI	Group A	Group B	Group C	Group D
Mean	1.15	1.02	1.67	1.75
SD	0.29	0.20	0.35	0.47
p value	< 0.001 Significant			

Table – 4 Comparison of mean probing pocket depth (in mm) values between group A, B, C and D

PPD	Group A	Group B	Group C	Group D
Mean	2.53	2.65	5.41	5.29
SD	0.62	0.61	0.51	0.47
p value	< 0.001 Significant			

Table – 5 Comparison of mean clinical attachment (in mm) loss between group A, B, C and D

CAL	Group A	Group B	Group C	Group D
Mean	2.59	2.65	5.35	5.41
SD	0.62	0.61	0.70	0.79
p value	< 0.001 Significant			

Table – 6 Mean salivary c reactive protein (mg/L) in group A, B, C and D

Salivary CRP (mg/L)	Group A	Group B	Group C	Group D
Mean	0.17	0.09	1.32	0.60

Table – 7 Correlation between salivary c reactive protein (mg/L) and clinical parameters of group A

Group A	**Correlation coefficient**	**Correlation**
Age Vs Salivary CRP	-0.239	Negative correlation
BMI Vs Salivary CRP	-0.193	Negative correlation
GI Vs Salivary CRP	0.474	Moderate correlation
PPD Vs Salivary CRP	0.71	Good correlation
CAL Vs Salivary CRP	0.663	Good correlation

Table – 8 Correlation between salivary c reactive protein (mg/L) and clinical parameters of group B

Group B	**Correlation coefficient**	**Correlation**
Age Vs Salivary CRP	-0.142	Negative. correlation
BMI Vs Salivary CRP	-0.179	Negative. correlation
GI Vs Salivary CRP	0.571	Moderate correlation
PPD Vs Salivary CRP	0.660	Good correlation
CAL Vs Salivary CRP	0.648	Good correlation

Table – 9 Correlation between Salivary c reactive protein (mg/L) and clinical parameters of group C

Group C	**Correlation coefficient**	**Correlation**
Age Vs Salivary CRP	-0.345	Negative. correlation
BMI Vs Salivary CRP	-0.255	Negative. correlation
GI Vs Salivary CRP	0.751	Good correlation
PPD Vs Salivary CRP	0.47	Moderate correlation
CAL Vs Salivary CRP	0.401	Moderate correlation

Table – 10 Correlation between Salivary CRP (mg/L) Vs Other parameters in Group D

Group D	**Correlation coefficient**	**Correlation**
Age Vs Salivary CRP	0.076	Very low correlation
BMI Vs Salivary CRP	0.561	Moderate Correlation
GI Vs Salivary CRP	0.749	Good correlation
PPD Vs Salivary CRP	0.691	Good correlation
CAL Vs Salivary CRP	0.86	Good correlation

Table – 11 A Comparison of distribution level of salivary c reactive protein values between groups

Salivary CRP (mg/L)	**Group A**	**Group B**	**Group C**	**Group D**
< 0.1	12	14	6	6
> 0.1	5	3	11	11

Table – 11 B p value comparison among group A to other groups

p value	Group A Vs Group C,D	0.039 Significant
	Group B Vs Group C	0.005 Significant
	Group B Vs Group D	0.005 Significant

TABLE -12 Distribution of delivery week of infant among the group A, B, C and D

Delivery week	**Group A**	**Group B**	**Group C**	**Group D**
> 37	14	6	8	6
33 - 37	3	10	6	10
≤ 32	0	1	3	1
p value	Group A Vs	0.019 Sig	0.06 NS	0.019 Sig
	0.034 Significant			

TABLE – 13 Distribution of birth weight (kilogram) among the group A, B, C and D

BIRTH WEIGHT	**Group A**	**Group B**	**Group C**	**Group D**
≤ 2.5	1	1	6	7
> 2.5	16	16	11	10
p value	Group A Vs	1.0 NS	0.085 NS	0.043 NS
p value	0.015 Significant			

Table- 14 Distribution of gestational week during sampling among the group A, B, C and D

Gestational week at sampling	**Group A**	**Group B**	**Group C**	**Group D**
< 20	12	12	12	8
21 – 28	3	3	4	7
> 28	2	2	1	2
	Group A Vs	1.0 NS	0.788 NS	0.301 NS
p value	0.670 Not Significant			

Table – 15 Correlation of delivery week, birth weight, gestational week with salivary CRP (mg/L) in group A

Group A	**Correlation coefficient**	**Correlation**
Delivery week Vs Salivary CRP	-0.557	Negative Moderate Correlation
Birth weight Vs Salivary CRP	-0.471	Negative Moderate Correlation
Gestational Week Vs Salivary CRP	0.258	Low correlation

Table – 16 Correlation of delivery week, birth weight, gestational week with salivary CRP (mg/L) in group B

Group B	**Correlation coefficient**	**Correlation**
Delivery weeks Vs Salivary CRP	0.044	Very low correlation
Birth weight Vs Salivary CRP	0.35	Low correlation
Gestational Weeks at sampling Vs Salivary CRP	0.309	Low correlation

Table -17 Correlation of delivery week, birth weight, gestational week with salivary CRP (mg/L) in group C

Group C	Correlation coefficient	Correlation
Delivery weeks Vs Salivary CRP	-0.818	Negative. Good correlation
Birth weight Vs Salivary CRP	-0.725	Negative. Good correlation
Gestational Weeks at sampling Vs Salivary CRP	0.017	Very Low correlation

Table -18 Correlation of delivery week, birth weight, gestational week with salivary CRP (mg/L) in group A

Group D	Correlation coefficient	Correlation
Delivery weeks Vs Salivary CRP	-0.706	Negative Good correlation
Birth weight Vs Salivary CRP	-0.663	Negative Good correlation
Gestational Weeks at sampling Vs Salivary CRP	0.132	Very Low correlation

Table 19: Odd ratio and confidence interval calculation between control group A and group B about low birth weight parameter.

GROUP	LBW-PRESENT	LBW-ABSENT
GROUP B	1	16
GROUP A	1	16

Odds ratio	**1.0000**
95 % CI:	0.0574 to 17.4121
z statistic	0.000
Significance level	P = 1.0000

Table 20: Odd ratio and confidence interval calculation between control group A and group B about pre term birth parameter.

GROUP	PTB-PRESENT	PTB-ABSENT
GROUP B	11	6
GROUP A	3	14

Odds ratio	**8.5556**
95 % CI:	1.7358 to 42.1700
z statistic	2.638
Significance level	P = 0.0083

Table 21: Odd ratio and confidence interval calculation between control group A and group C about low birth weight parameter.

GROUP	LBW-PRESENT	LBW-ABSENT
GROUP C	9	8
GROUP A	1	16

Odds ratio	**15.7500**
95 % CI:	**1.674 to148.124**
z statistic	**2.411**
Significance level	**0.0159**

Table 22: Odd ratio and confidence interval calculation between control group A and group C about pre term birth parameter.

GROUP	PTB-PRESENT	PTB-ABSENT
GROUP C	6	11
GROUP A	3	14

Odds ratio	**2.5455**
95 % CI:	**0.516 to 12.546**
z statistic	**1.148**
Significance level	**0.2510**

Table 23: Odd ratio and confidence interval calculation between control group A and group D about low birth weight parameter

GROUP	LBW-PRESENT	LBW-ABSENT
GROUP D	7	10
GROUP A	1	16

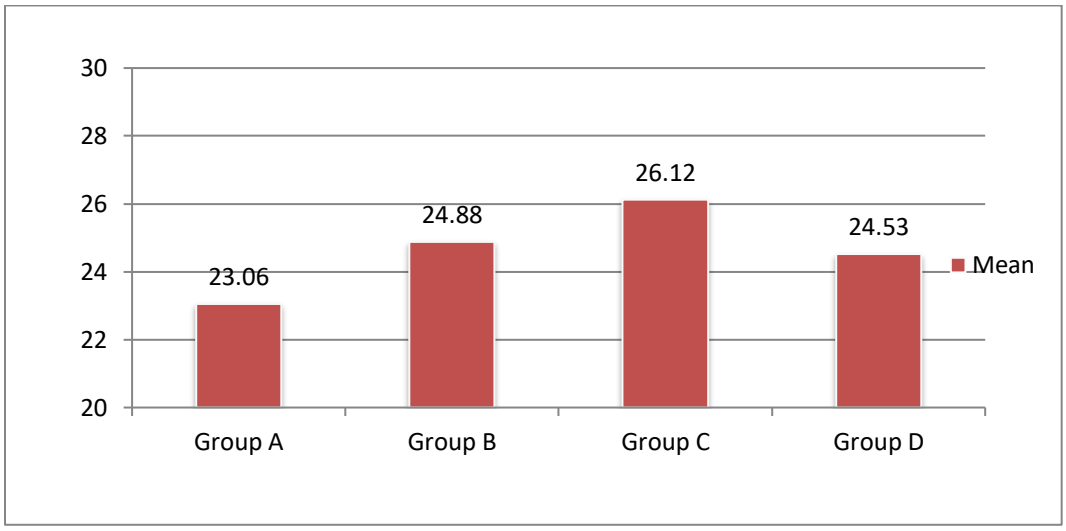
Odds ratio	**11.2000**
95 % CI:	1.1931 to 105.1363
z statistic	2.115
Significance level	P = 0.0345

Table 24: Odd ratio and confidence interval calculation between control group A and group D about pre term birth parameter.

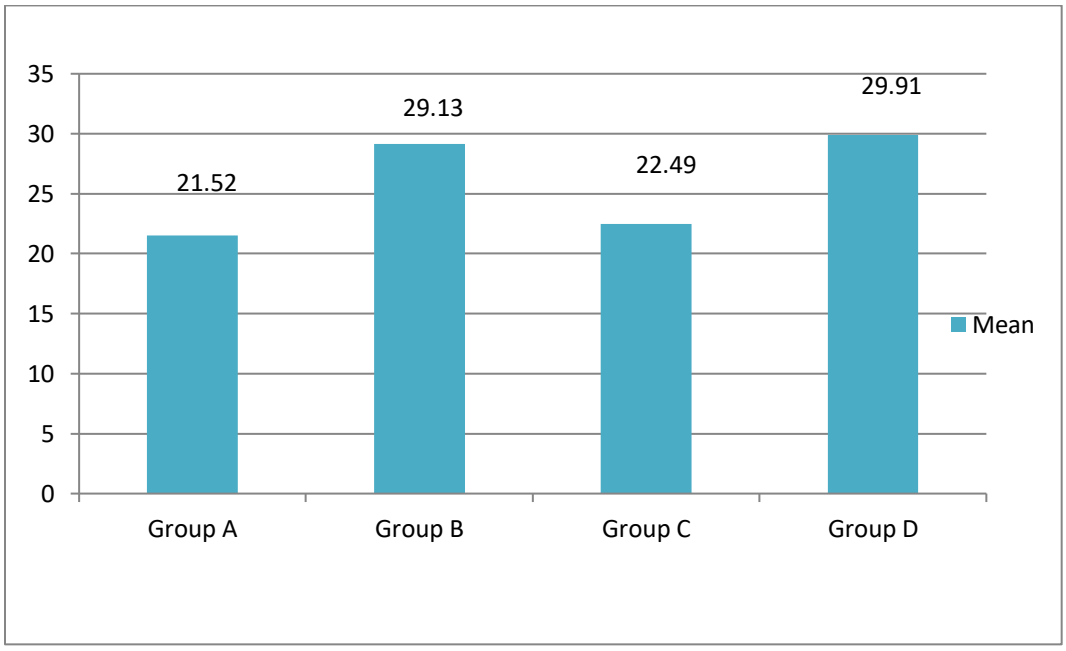
GROUP	PTB-PRESENT	PTB-ABSENT
GROUP D	11	6
GROUP A	3	14

Odds ratio	**8.5556**
95 % CI:	1.7358 to 42.1700
z statistic	2.638
Significance level	P = 0.0083

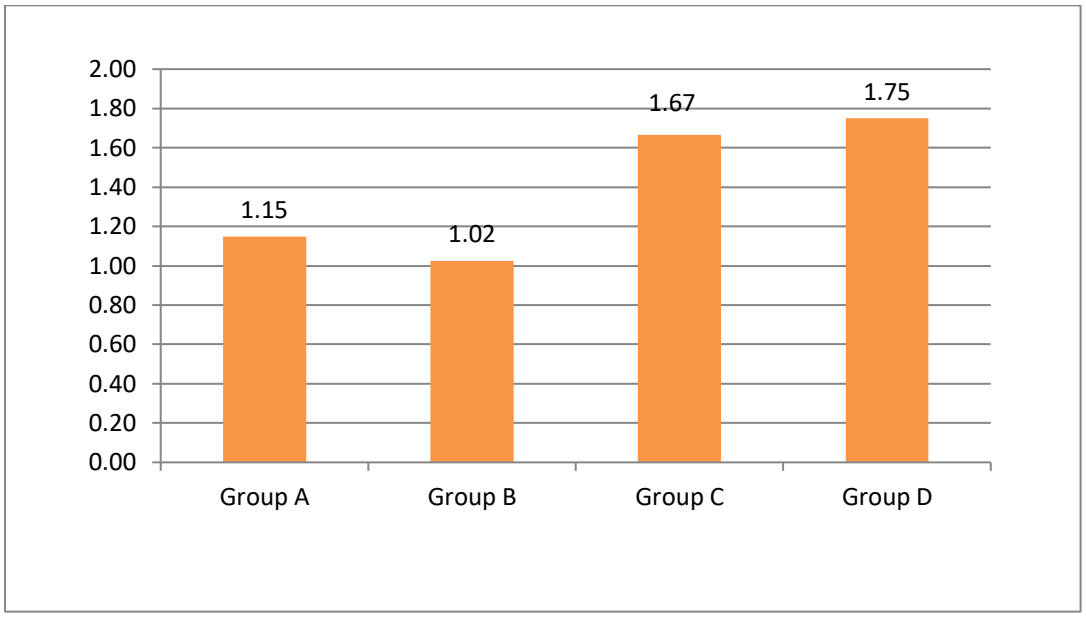
Graph -1 Comparison of mean age between group A, B, C and D.



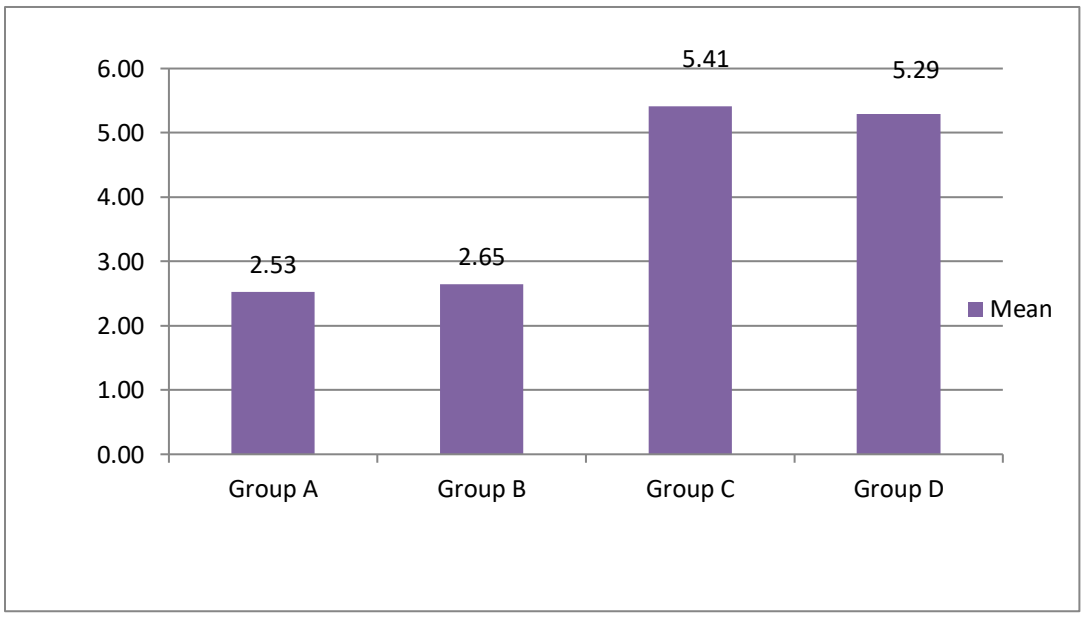
Graph -2 Comparisons of body mass index values (kg/m^2) between group A, B, C and D



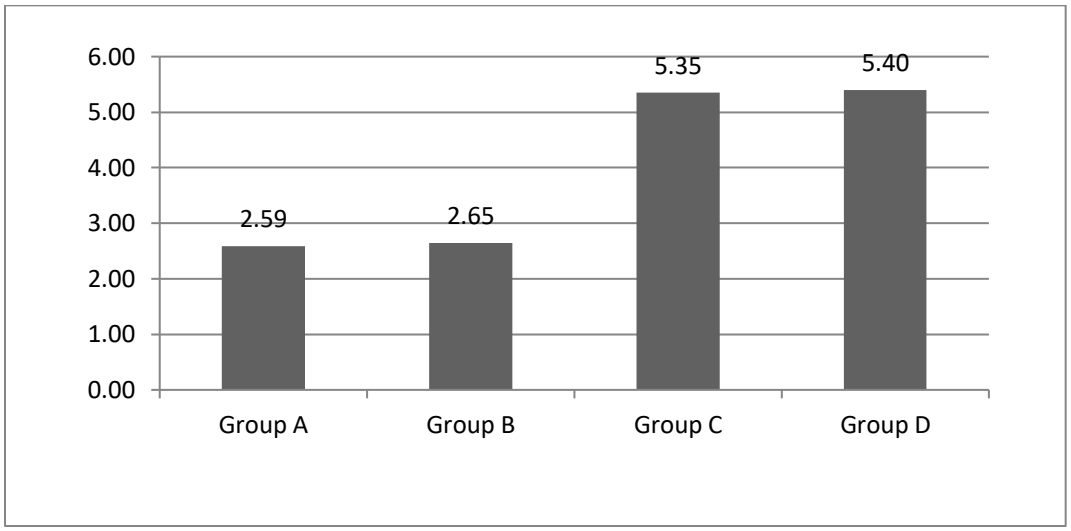
Graph -3 Comparison of Mean values of gingival index between the group A, B, C and D



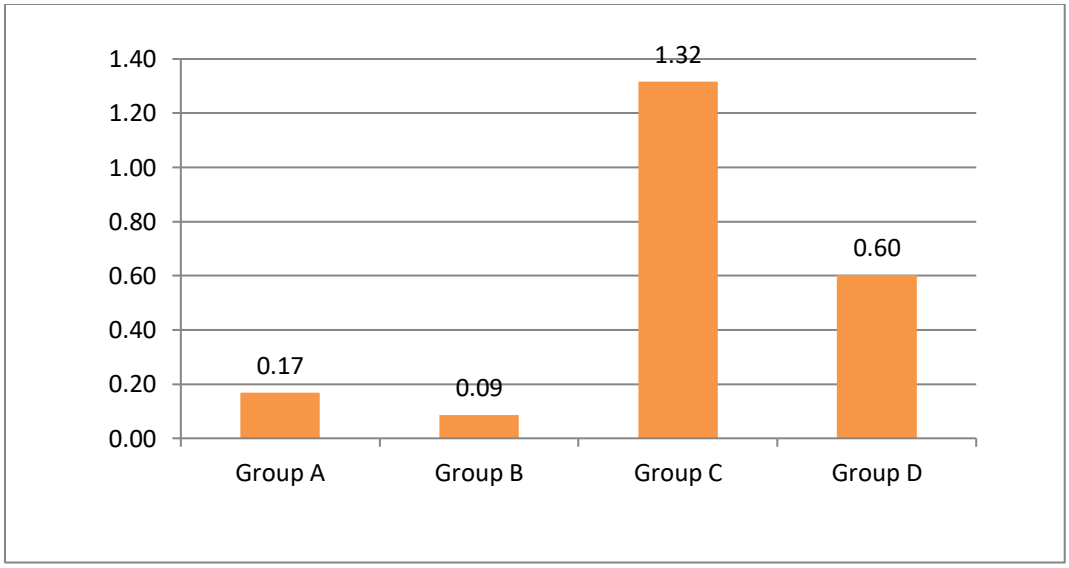
Graph – 4 Comparison of mean probing pocket depth (in mm) values between group A, B, C and D



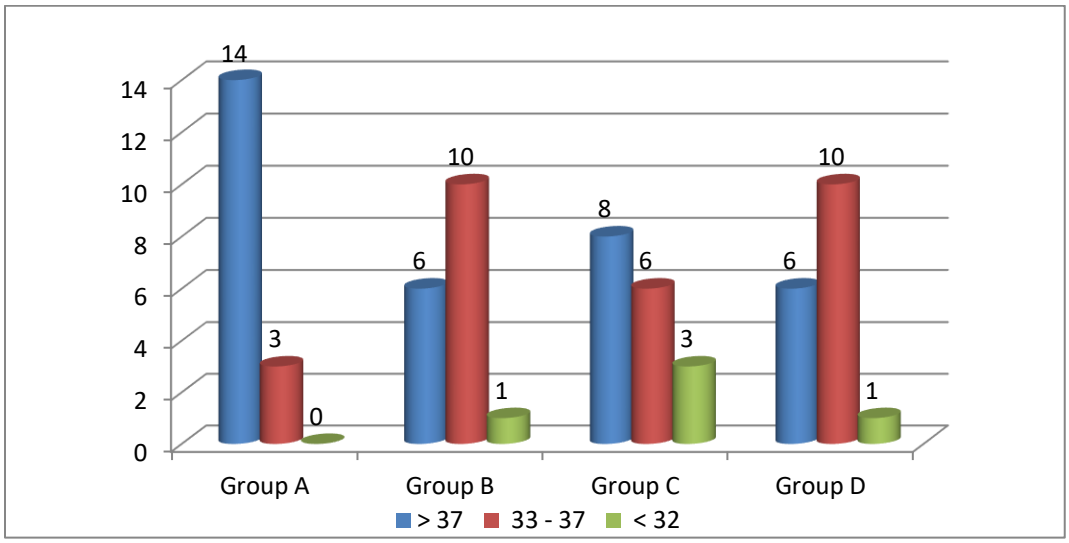
Graph-5 Comparison of mean clinical attachment (in mm) loss between group A, B, C and D



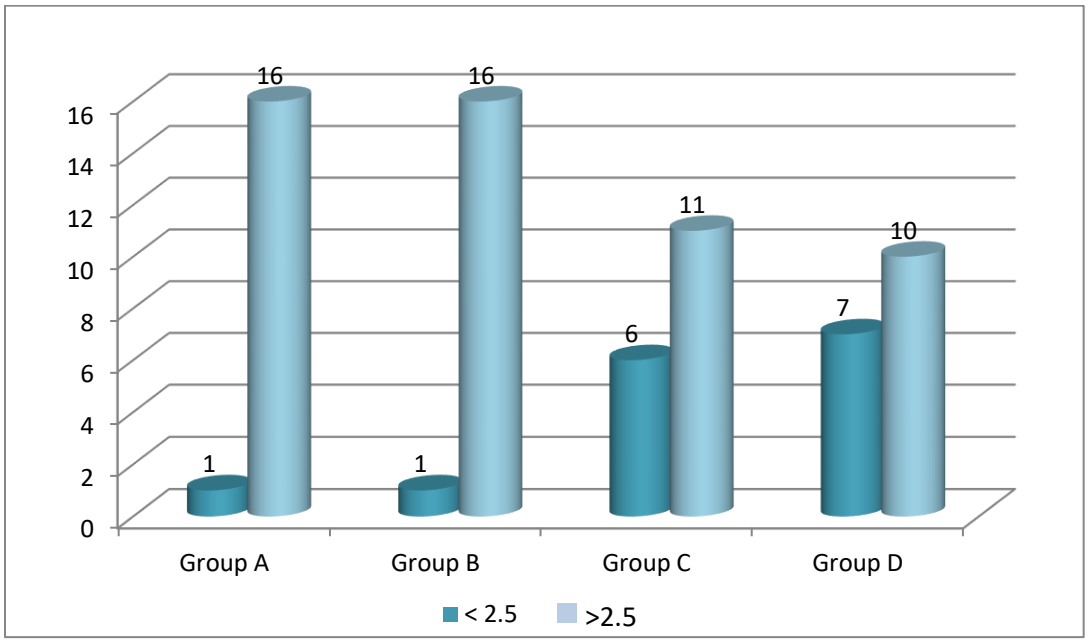
Graph-6 Mean salivary c reactive protein in group A, B, C and D



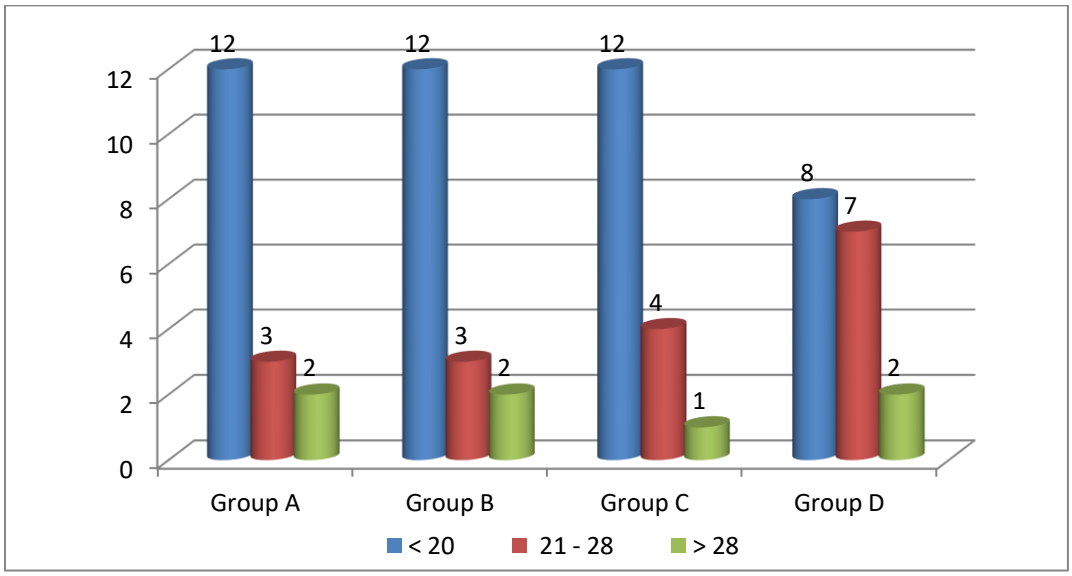
Graph – 7 Distribution of delivery week of infant among the group A, B, C and D



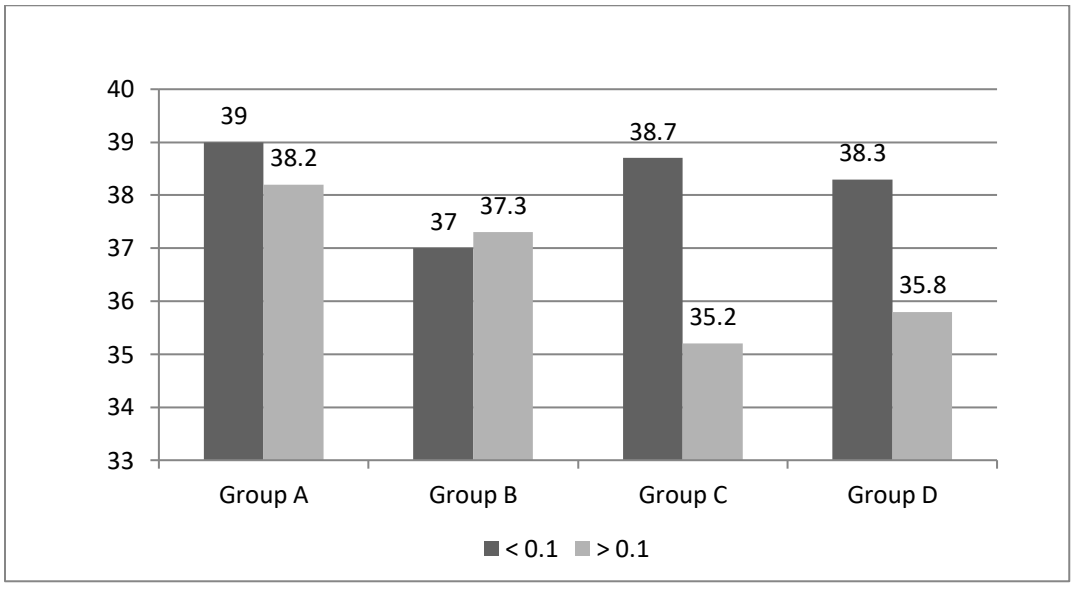
Graph -8 Distribution of birth weight (kilogram) among the group A, B, C and D



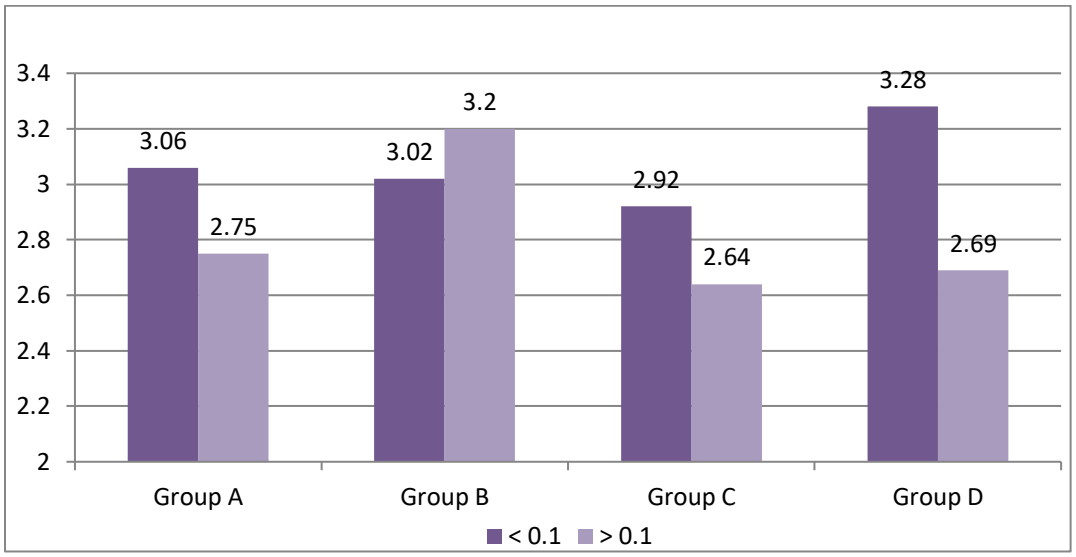
Graph – 9 Distribution of gestational week during sampling among the group A, B, C and D



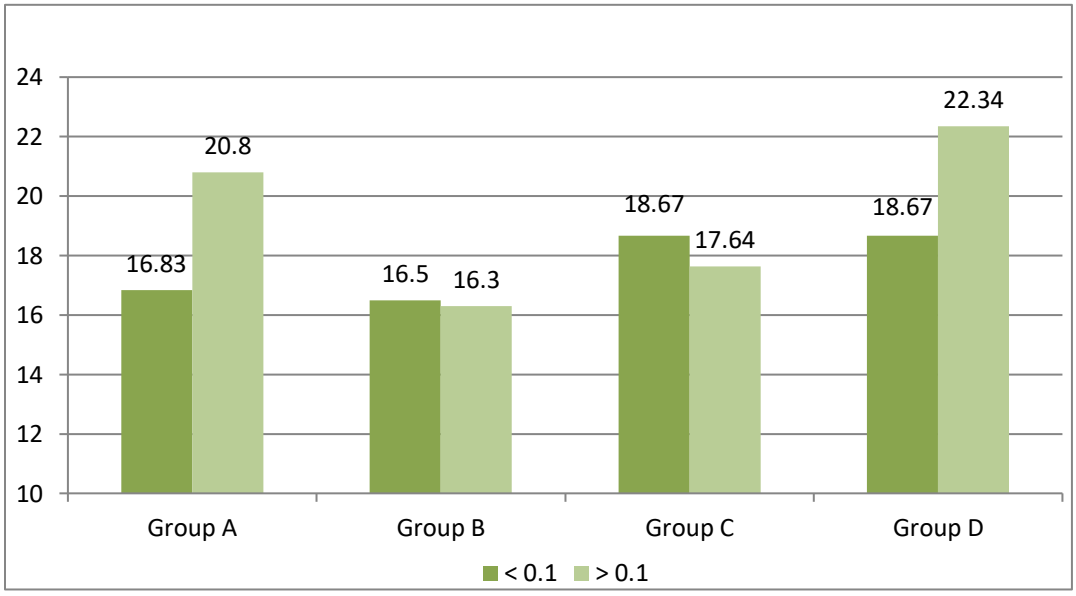
Graph – 10 Relationship between salivary CRP (mg/L) and delivery weeks.



Graph -11 Relationship between salivary CRP (mg/L) and birth weight.



Graph – 12 Relationship between salivary CRP (mg/L) and gestational week



DISCUSSION

A woman who lack their periodontal care during the initial period of their life, was the most important significant predictor of lacking their care during pregnancy period. [74] Changes in the oral cavity can occur with pregnancy, which include alterations in both the hard and soft tissues structures. [75] The periodontal changes such as pocket formation, increased tooth mobility and loss of attachment may lead to decrease in oral health status and leads to loss of teeth. [76,77] A multi-state study had concluded that, besides neglecting medical care during pregnancy, most of the expectant females of all ages do not seek for the dental care, even if the 50 percent of them have a dental problem. [78] Saliva is a unique biological fluid, which has an important role in the maintaining of oral physiology. It plays the major role in the process of oral and general health maintenance **(Humphrey 2001)** [79]. According to recent data, it mirrors general health condition which can reflect various systemic changes, which occurs in the body.

Serum CRP concentration usually increases in the case of systemic inflammatory conditions like systemic infection and inflammation. CRP was the most commonly used acute phase protein in the clinical practice. Few studies evaluated about the CRP levels in oral cavity. A study evaluated among 45 adults in whole saliva to detect the level of C reactive protein. The concentrations range from 0 to 472 pg/ml were found in samples. The level was higher in patients with gingivitis, moderate and severe periodontitis **(Pederson, 1995)** [80].

The aim of the study was to estimate the level of salivary c reactive protein in pregnant women with obesity and periodontitis and to identify their potential risk for

preterm birth. In this study, there were 4 groups based on obesity and periodontitis. The obesity was calculated using body mass index and periodontitis was considered when subject had 2 or more interproximal sites with CAL≥ 4mm that were not on the same tooth. (M4, M5, 1).

The mean body mass index of the sample were found to be increased in group B and D, which were 29.13 and 30.37 respectively , these results found to be in correlation with study done by **Naidoo T et al 2012** [81] . When the mean value of GI, PPD, CAL were compared. The values were increased in group C and D when compared to other groups. The values were 1.67 and 1.75, 5.41 and 5.29, 5.35 and 5.41 respectively. The mean salivary c reactive protein level in group A, B, C, and D were 0.17, 0.09, 1.32 and 0.60 respectively. The mean level of salivary c reactive protein were comparatively increased in group with periodontitis, both periodontitis and obesity when compared to other. The increased mean level of salivary c reactive protein found correlated with the study done by **P.Savitha and R.Shasmitha in 2015.(r37).**

The correlation between body mass index to salivary c reactive protein was correlated in all four groups, while moderate correlation was found in group D. The moderate and good correlation between gingival index and salivary CRP was seen in group A and B and in group C and D respectively. The PPD and CAL was found to have good correlation with salivary C reactive protein in group A, B and D and moderately correlated in group C.

Pre term birth was defined as delivery at <37 weeks of gestation [1]. In group A, 3 subject had preterm birth, while in group B, C and D 11, 9, and 11 preterm birth

were recorded respectively. Statistically significance were found between group A when compared B,C and D. In this study, subjects with periodontitis had increased risk of preterm birth, the similar results were found in the study done by **Agueda et al (2008)** [3]**, Macedo et al (2014)** [82] **and Pitiphat W et al (2008)** [48].

Infants weighing less than 2,500 grams at birth are considered to be low birth weight. In group D, 7 subjects gave birth to infant with weight ≤2.5kg which was considered as low term birth, found to be statistically significant when compared to group A respectively. **Offenbacher S et al (2001)**[39] **, Pederson, E (1995)**[80] **, Ide et al (2013)** [83] found that patient with periodontitis gave delivery to low birth weight infants which was found to be correlating with results of the present study. When infant weight and salivary CRP are correlated, in group C and D they found to have negative good correlation ie., when salivary c reactive protein level increased there were increased risk for preterm birth (decreased delivery weeks).

The association was stronger in obesity patient to get preterm birth with odds ratio=8.556 and C.I = 1.7358 to 42.1700. The association was stronger in group C to get low birth weight infants and preterm birth with odds ratio= 15.75 , C.I = 1.67 to 148.12 and odds ratio= 2.54 , C.I = 0.516 to 12.546 respectively. The association was also stronger in group D patients to get preterm birth and low birth weight with odds ratio=11.2000,C.I= 1.1931 to 105.1363 and odds ratio=8.5556, C.I= 1.7358 to 42.1700 respectively. The odds ratio of more than one value suggest , there is strong association between them. [2] (C.I- confidence interval)

Periodontal status may be associated with PTB and PE **(Boggess et al. 2003)** [84] . The association between maternal periodontal health and adverse pregnancy

outcomes has been confirmed out in many studies. Our findings were consistent with the results from some of the previous studies conducted. **Offenbacher 1991** suggested that the mechanism feasibly between the periodontal inflammation and the poor foetal growth includes the translocation of periodontal pathogens, inflammatory mediators, and bacterial products such as lipopolysaccharides to the fetoplacental unit.

Obesity is also one of the largest attributable and potentially modifiable risk factors for PE and PTB **(Torloni et al. 2009)** [85]. **Cnattingius et al. (2013)** [86] reported that maternal overweight and obesity were associated for an increased risk for PTB. Maternal obesity is associated with inflammatory up-regulation through increased production of adipokines and enhanced systemic secretion of proinflammatory cytokines **(Ramsay et al. 2002)** [87] . Specific complications of pregnancy that occur in obesity can also lead to PTB **(Parker et al. 2014)** [88] ; the potential mediators of pre-pregnancy obesity and PTB are hypertensive disorders of pregnancy, chorioamnionitis, gestational diabetes, and other common conditions among obese women . However, **Ehrenberg et al. (2009)** [89] found opposing results between maternal obesity and spontaneous PTB, and explained that the results may be due to chance caused by the small sample size or their definition of preterm birth. We also found that there was low correlation between obesity subjects, which was consistent with above study results. Considering the abundance of evidence supporting the association among obesity, periodontitis, and PTB, it can be inferred that obesity and periodontitis before or during pregnancy have synergistic effects on PTB. There is a link between obesity and periodontal disease, but the risk factors that aggravate these diseases should be clarified to clarify the meaning of this association. Despite the growing volume of data generated by human studies and the experimental

animal models, the clinical application of this information to the practice of dentistry needs to be carefully delineated. Although most of the studies to date indicate a positive correlation, it is still too early to attribute a cause and effect.

In an overview of periodontitis and obesity, whether one condition stands as a risk factor for another or whether one disease causes another has yet to be elucidated. What has emerged from the literature, however, is that an association between obesity and periodontitis exists and that association most likely lies in the commonality of their inflammatory pathways. The relentless release of pro-inflammatory cytokines into the systemic circulation from adipose tissue in obese individuals provides a systemic inflammatory overload. These cytokines may directly injure the periodontal tissues or reduce the vascular blood flow to the periodontal tissues. Periodontitis, itself an inflammatory disease, induces its own set of cytokines both locally and systemically in response to bacterial pathogens and endotoxins and thus adds to the level of circulating proinflammatory mediators. As such, periodontitis increases the systemic level of inflammation and thus may contribute to systemic inflammatory diseases and a bidirectional link. Long-term prospective studies are needed to further elucidate specific cause and effect relationships between obesity and periodontitis as well as to the wider body of other chronic inflammatory diseases.

The limitation of the study was those patients were requited from a single clinic. The study was only observational type. The patient did not undergo any periodontal treatment measures. The pre-pregnancy weight details of the patient was obtained only face to face interview, as there can be sometime a subjective variance. Overweight and obesity were combined into a single category for analysis because of the

small number of obese pregnant women. Within the limitations of the study, it is shown that the obesity and periodontitis act synergistically risk factor for preterm birth. In future, the studies should be directed with treatment measures. The salivary C reactive protein act as a reliable method to estimate the level of inflammation, which is a non invasive and simple method. Anticipatory guidance should be given to women with obesity to maintain proper periodontal health status to avoid a risk factor for preterm birth in future. The awareness about periodontal health maintenance and risk poor oral hygiene status should be increased among the public.

SUMMARY AND CONCLUSION

The present study was conducted to estimate the level of salivary C reactive protein in pregnant women with obesity and periodontitis and to find their risk for preterm birth. A total of 74 pregnant women were included in the study. Depending on inclusion and exclusion criteria the sample was divided into 4 groups equally based on obesity and periodontitis criteria. Due to loss of follow up 6 patients were excluded from the study.

The clinical parameters like Gingival index, PPD, CAL, and BMI were recorded. Salivary c reactive protein was analyzed using particle enhanced turbidimetric immunoassay. The patient were followed and observed for post obstetrics details.

From the results of the study, we arrive at the following conclusions:

- In patients with only obesity, association was found between pre term birth and obesity
- In patients with only periodontitis, association was found between both preterm birth and low birth weight of the infants.
- In patient with both obesity and periodontitis, association was found between them and preterm birth and low birth weight.
- Obesity and periodontitis are strongly associated and act as a risk for preterm birth of low birth weight.
- Salivary C reactive protein acts as a reliable non invasive way to evaluate the inflammatory levels.

- The results of this study indicate that pregnant women with both obesity and periodontitis are significantly more likely to have PTB than pregnant women in control group. Our results support that maternal obesity must be controlled and managed prior to pregnancy; in particular, obese pregnant women must manage and maintain better periodontal health than normal weight pregnant women in order to reduce PTB with LBW.

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ANNEXURE 1

INSTITUTIONAL ETHICAL COMMITTEE

Best Dental Science College and Hospital
Ultra Nagar, Madurai - 625 104.
RECOGNIZED BY DENTAL COUNCIL OF INDIA, NEW DELHI
AFFLIATED TO THE TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY, CHENNAI)

IRB/IEC Reference No: 2016-STU-BrII-SUH-21

Project title: Estimation of Salivary C Reactive Protein in pregnant women with Obesity and Periodontitis and their potential risk for Preterm Birth

Principal Investigator: Dr. T.Suganya Harshni, PG student

Review: New/Revised/Expedited

Date of Review: 27/09/2016

Date of previous review, if revised application:

Decision of the IEC/IRB:

- Provisional approval to conduct the study is being given
- The results of this study, along with summary are to be submitted for obtaining final approval

Recommended time period: one year (28-09-17)

PRINCIPAL
BEST DENTAL SCIENCE COLLEGE
MADURAI-625104

ETHICS COMMITTEE SIGN DATE BDSC

INSTITUTIONAL REVIEW BOARD SIGN. DATE BDSC

Signature of Member Secretary

NB:

- **Inform IRB/IEC immediately in case of any issue(s)/adverse events**
- **Inform IRB/IEC in case of any change of study procedure, site and investigator**
- **This permission is only for the period mentioned above**
- **Annual report to be submitted to IEC/IRB**
- **Members of IEC/IRB have right to monitor the trail with prior intimation**

ANNEXURE 2

INFORMED CONSENT FORM

Name: Mrs	**REF.No:**
Address:	**SEX : Female**
AGE: Years	

I ……., exercising my free power of choice, hereby give my consent to be included as a participant in the study .

I agree to the following;

1. I have been informed to my satisfaction about the purpose of the study
2. I understand that the study involves questions which may sometimes be personal
3. I agree to co -operate fully for complete examination
4. I agree to report to my doctor for a regular follow up and when required for the research
5. I hereby give permission to use my medical records for research purpose. I have been told that the investigating doctor and the institution will keep my identity confidential
6. I understand that I have rights to withdraw myself from the study and also that the investigator has the right to exclude me from the research at any point of time.

INVESTIGATOR:

SIGNATURE OF THE PARTICIPANT: Signature/Thumb impression

ANNEXURE 3

ஒப்புதல் படிவம்

திரு. / திருமதி ..... வயது .....

தற்பொழுது .....

என்ற முகவரியில் வசித்து வரும் நான் **முழுசுயநினைவுடனும் மனப்பூர்வமாகவும்** யாருடைய தூண்டுதலின்பெயரில் அல்லாமலும் உறுதி கூறுவது என்னவென்றால்,

1. என்னுடைய உமிழ்நீரை மருத்துவ சோதனை மாதிரிக்காக நான் அளிக்கின்றேன்.
2. செயல்முறையினை **எனக்கு நன்கு விளக்கப்பட்டுள்ளது.** மேலும் இதில் வரும் நன்மை தீமையினை என் **சுயநினைவோடு நான் புரிந்து கொண்டேன்.** இதன் மூலம் என்னுடைய மனப்பூர்வமான சம்மதத்தை நான் உறுதிப்படுத்துகிறேன்.

இடம்:

நாள் :

கையொப்பம்

ANNEXURE 4

BEST DENTAL SCIENCE COLLEGE, MADURAI

TAMILNADU DR.MGR MEDICAL UNIVERSITY

DEPARTMENT OF PERIODONTOLOGY

MAIN DISSERTATION PROFORMA

PATIENT INFORMATION

REFERENCE NUMBER: S.NO: DATE:

NAME OF THE PATIENT: AGE: SEX:
ADDRESS:

CONTACT NUMBER:

OBSTETRICS INFORMATION

GESTATIONAL AGE:

AGE AT FIRST DELIVERY:

PARITY:

NONE:

ONE OR MORE:

HISTORY OF ABORTION: YES / NO

HISTORY OF PRETERM BIRTH: YES / NO

HISTORY OF SCALING DONE BEFORE ONE YEAR: YES / NO

SMOKING BEFORE PREGNANCY: YES / NO

WEEKLY DRINKING BEFORE PREGNANCY: NO / LESS THAN 1 / ONCE OR MORE

WEEKLY EXERCISE BEFORE PREGNANCY : NO / 1-2 TIMES / MORE THAN 3 TIMES

WEIGHT BEFORE PREGNANCY: WEIGHT AT PRESENT:

HEIGHT:

FAMILY HISTORY:

MEDICAL HISTORY:

SYSTEMIC DISEASE:

SYSTEMIC MEDICATION:

GINGIVAL INDEX – LOE AND SILNESS 1963

B														
P														
	17	16	15	14	13	12	11	21	22	23	24	25	26	27

B														
L														
	47	46	45	44	43	42	41	31	32	33	34	35	36	37

CALCULATION :

IMPRESSION :

PROBING POCKET DEPTH

B														
P														
	17	16	15	14	13	12	11	21	22	23	24	25	26	27
	47	46	45	44	43	42	41	31	32	33	34	35	36	37
L														
B														

IMPRESSION :

CLINICAL ATTACHMENT LOSS

B														
P														
	17	16	15	14	13	12	11	21	22	23	24	25	26	27
	47	46	45	44	43	42	41	31	32	33	34	35	36	37
L														
B														

IMPRESSION :

PATIENT HAS PERIODONTITIS ACCORDING TO CRITERIA : YES / NO

BMI CALCULATION: $\frac{\text{WEIGHT IN KILOGRAM}}{(\text{HEIGHT IN METERS})^2}$

IMPRESSION FROM BMI:

PATIENT INCLUDED UNDER GROUP:

GROUP A: NEITHER PERIODONTITIS NOR OBESITY

GROUP B: ONLY OBESITY

GROUP C: ONLY PERIODONTITIS

GROUP D: BOTH OBESITY AND PERIODONTITIS

SALIVARY C- REACTIVE PROTEIN REPORT:

POST PARTUM DETAILS

DELIVERY OF INFANT : WEEKS

PRETERM BIRTH: YES / NO

DATE OF DELIVERY:

WEIGHT OF INFANT:

SIGNATURE OF THE INVESTIGATOR SIGNATURE OF THE GUIDE

MASTER CHART

GROUP A

REF.NO	AGE	BMI	GI	PPD	CAL	SALIVARY CRP	d.weeks	weight	g.weeks
A1	33	22	0.85	2	2	<0.1	38	2.9	20
A2	22	19.7	1.65	3	3	<0.1	37	2.8	24
A3	19	20.88	1.14	2	3	<0.1	40	3.4	8
A4	24	23.45	0.81	2	2	<0.1	40	2.7	20
A5	21	22.26	0.93	2	2	<0.1	38	2.8	18
A6	28	22.1	1.07	2	2	<0.1	40	3	16
A7	27	19.97	1.03	3	3	<0.1	39	3.4	32
A8	21	20.2	1	3	3	0.1	40	2.8	18
A9	20	20.45	1.75	4	4	1.1	36	2.45	24
A10	20	24.8	1.15	3	3	0.1	39	3.3	20
A11	24	24	0.77	2	2	<0.1	39	3	8
A12	20	18.17	1.05	2	2	<0.1	40	3.2	24
A13	20	22.31	1.16	2	3	<0.1	38	2.9	12
A14	24	23.62	1.3	3	2	0.1	37	2.6	12
A15	22	18.32	1.09	3	3	<0.1	40	3.2	8
A16	23	20.82	1.66	3	3	0.2	39	2.6	30
A17	24	23.24	1.13	2	2	<0.1	39	3.4	12

GROUP B

REF.NO	AGE	BMI	GI	PPD	CAL	SALIVARY CRP	d.weeks	weight	g.weeks
B1	28	25.8	1.15	3	3	0.1	38	3	7
B2	28	25.72	1.41	3	3	<0.1	39	2.7	16
B3	20	25.8	0.74	3	3	<0.1	40	3	20
B4	24	27.39	0.82	2	2	<0.1	36	2.6	8
B5	26	25.33	0.9	2	2	<0.1	40	2.95	8
B6	22	28.14	1.1	3	2	0.1	37	3.2	14
B7	20	34.89	1.13	2	2	<0.1	34	3	7
B8	24	30.46	1.12	2	3	<0.1	36	3.2	20
B9	29	28.06	0.79	2	2	<0.1	36	2.9	7
B10	30	31.73	0.91	3	3	<0.1	36	3.2	32
B11	35	34.13	0.98	3	3	<0.1	32	2.5	28
B12	28	32.79	0.84	3	3	<0.1	37	3	9
B13	23	27.83	1.36	4	4	0.4	37	3.4	28
B14	20	33.72	1.09	2	2	<0.1	35	3	32
B15	24	27.04	0.84	3	3	<0.1	37	3.44	24
B16	20	31.2	1.18	3	3	<0.1	40	3.4	12
B17	22	25.23	1.04	2	2	<0.1	40	3.4	8

GROUP C

REF.NO	AGE	BMI	GI	PPD	CAL	SALIVARY CRP	d.weeks	weight	g.weeks
C1	27	22.37	1.52	5	5	<0.1	38	3.2	12
C2	30	24.79	1.5	6	6	0.1	39	3.2	17
C3	25	23.71	1.32	5	5	<0.1	37	3	32
C4	29	24.16	1.64	5	5	<0.1	39	3	20
C5	24	20.44	1.42	6	6	0.2	36	2.5	24
C6	27	21.003	1.58	5	5	<0.1	40	2.6	8
C7	26	23.922	2.3	6	6	6.8	32	2.4	17
C8	22	22.47	1.3	5	4	<0.1	40	2.7	12
C9	27	20.44	2.2	6	6	0.6	36	2.6	24
C10	28	24	1.36	5	5	0.1	39	3.2	16
C11	34	20.08	1.92	5	6	0.3	37	2.6	24
C12	25	24	1.9	6	6	0.6	35	2.5	8
C13	21	24.21	1.44	5	4	<0.1	38	3	28
C14	19	22.31	1.49	5	5	0.8	34	2.5	8
C15	35	22.66	1.49	5	5	0.1	39	3.2	20
C16	20	20	2.4	6	6	11.2	28	1.8	20
C17	25	21.77	1.55	6	6	1.2	32	2.4	16

GROUP D

REF.NO	AGE	BMI	GI	PPD	CAL	SALIVARY CRP	D.weeks	weight	g.weeks
D1	21	25.55	1.38	5	5	0.3	36	2.4	12
D2	31	31.11	1.45	5	5	0.2	36	2.5	18
D3	26	29.13	1.53	5	5	0.1	37	3.2	21
D4	27	36.2	2.8	6	8	4.8	32	2.2	24
D5	22	31.34	1.464	5	5	<0.1	39	3.2	12
D6	24	32.65	2.7	6	6	1.5	35	2.5	20
D7	25	25.1	1.79	5	5	<0.1	40	3.4	12
D8	20	30.53	2.12	6	5	0.8	35	2.5	32
D9	24	29.5	1.93	5	5	0.1	40	3.2	24
D10	28	32.1915	1.41	5	5	0.1	38	3.2	24
D11	21	27.77	1.9	5	6	<0.1	40	3.2	28
D12	22	25.51	1.43	5	5	<0.1	39	3.5	28
D13	25	27.27	1.18	5	6	<0.1	36	3.2	20
D14	34	29.75	1.68	5	5	<0.1	36	3.2	12
D15	25	28.571	1.36	6	5	0.9	35	2.5	12
D16	22	30.6	2.2	6	6	1.1	34	2.4	28
D17	20	35.714	1.41	5	5	0.1	36	2.8	31